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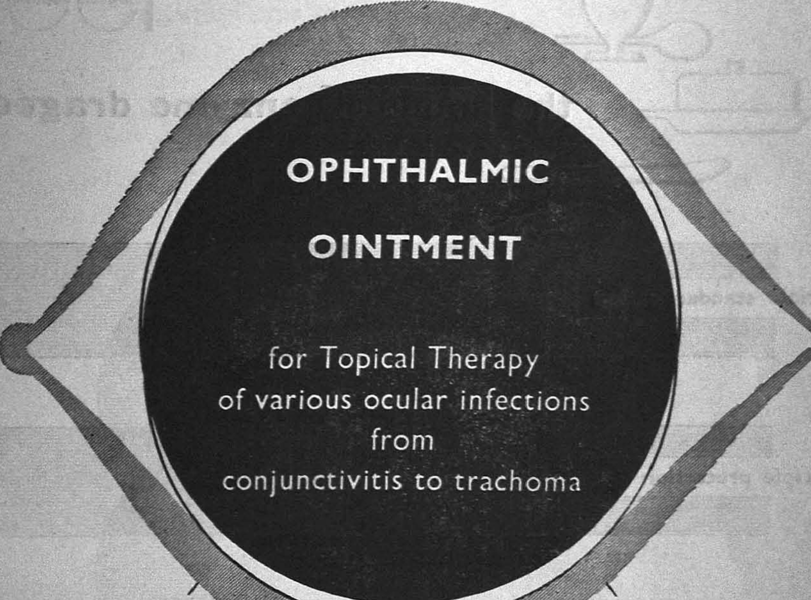

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—EDITOR.

EDITORIAL

WE are doubly indebted to the President of our Association for his untiring, unflagging and ever increasing interest he has shown in the cause and for the good of the Service Personnel, in more than one way. If this Number has seen the light of day even though it be belated in its appearance, it is due wholly to him. At the start it seemed as if there were very little material but very soon the contributions overflowed the target fixed and much of the excess has been reserved for the next issue.

INTEGRATED COURSE—THE NEED OF THE HOUR

Whilst it is very essential to have specialists in different branches of medical sciences and to this end, there shall be an Integrated School of Post-Graduate Medical Education, for the pursuit of such specialist courses of study, it cannot be denied that the general population of our country to-day needs numerically, not merely the high grade specialists, but a vast army of general practitioner cadre who have to combine in their accomplishments the peculiar blend of the essentials of medicine, surgery and midwifery, especially needed, to cater to the rural population. The man or the woman on the initial rung of the service ladder posted to the rural parts—say—the taluq headquarters hospital—having under his or her territorial jurisdiction, a cluster of villages must be capable of adaptation and adjustment to apply the knowledge and skill gained during the undergraduate period of training. Despite the fact that the general medical qualification denotes proficiency, not in medicine alone, nor in surgery but that it is an all round qualification, yet after acquiring the university

degree and undergoing the internship and house-surgeoncy subsequently, the pioneering medico in the inaugural phase of his service career in the mofussil may find himself helpless, unprepared, and entertain a frustrated outlook ; in short he may seem totally 'not attuned' to the specific need of the core of the country. Frankly speaking, the young service officer for all practical purposes misses something with which to grapple or shoulder the responsibility thrust on or expected of him. This may be because of the atmosphere in large hospitals where when a patient's complaint is found to involve a particular part of the human anatomy, the patient concerned is immediately directed to the relevant section ; *e.g.*, for pain in the ear the patient is instructed to attend the oto-rhino-laryngologist ; if the conjunctiva be inflamed, he is sent away to the ophthalmic physician ; seeing a swollen joint in an individual, an immediate reference is made to the orthopaedic specialist and so on. It is indeed justifiable that for such prompting symptoms as have been catalogued, the different patients should be dealt with in the specific sections to the advantage of either. But when placed in the singular situation as in the distant rural areas where the recently recruited service officer has to act the role of all the specialists in himself, at once, under a different climate, it may seem a difficult proposition for him to tackle all and sundry problems that crop up day after day in his service career satisfactorily. It is to overcome this embarrassing predicament that it is suggested that a preparatory training may be given to a sufficiency of post-graduates in the art of general practitioners. It has been brought to our notice that such a course of training is available elsewhere and it is our fond hope that a similar provision may be made available at the post-graduate school envisaged, in order that we might be able to turn out the requisite number of qualified medical graduates fitted with the mantle of general practitioners.

Though disintegrated in a sense during the moulting phases of the different disciplines in the undergraduate period, yet by the integrated post-graduate course contemplated the post graduates may equip themselves appropriately to meet the challenge of the hour for the good of our country.

P. N. Rangiah.

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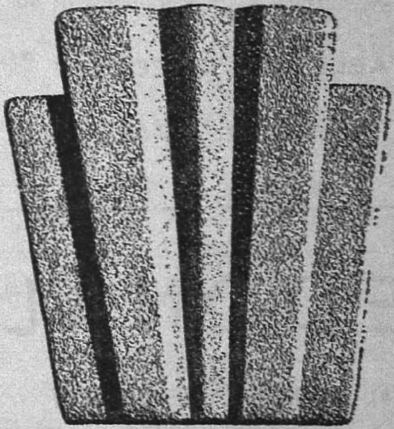

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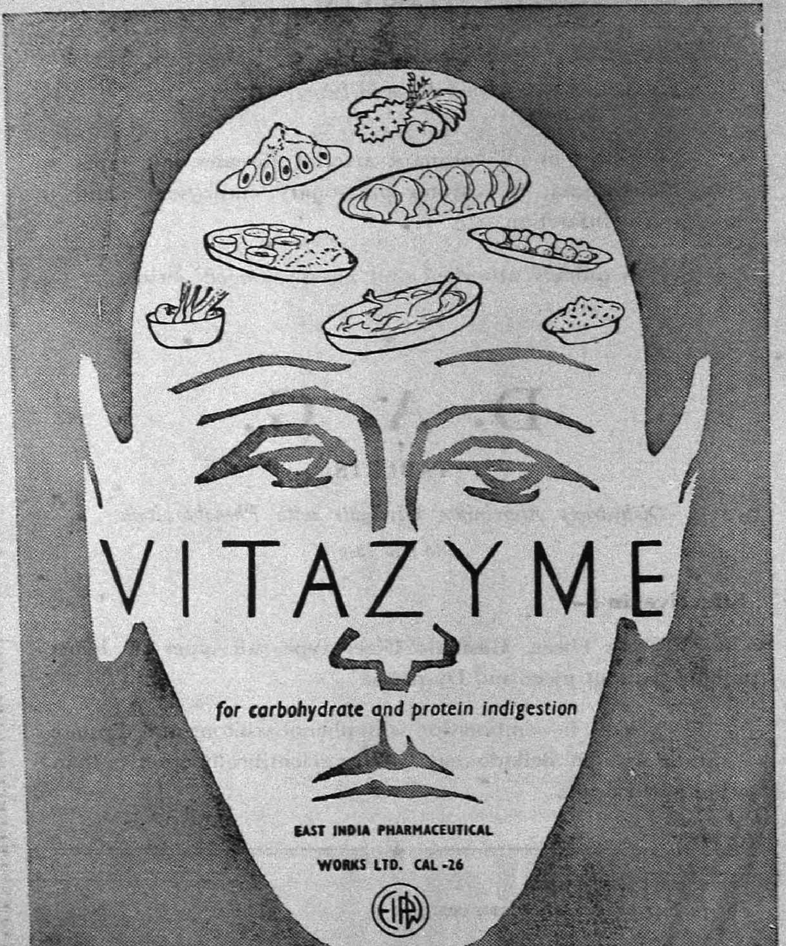
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Hepatic Coma or Cholaemia

Dr. R. SUBRAMANIAM*

M.D., M.R.C.P. (Lond.)

Just as uraemia represents the end stage of renal failure, cholaemia indicates liver failure. Cholaemia is often seen as a terminal manifestation of cirrhosis liver. It may also occur in the course of infective hepatitis, toxaemias of pregnancy, and acute yellow atrophy. Irrespective of the causes, the clinical picture is the same. In all these conditions, the liver is damaged. This brief article deals with cholaemia that occurs in the course of cirrhosis liver. In 1958-59, we had twelve cases of hepatic coma secondary to cirrhosis liver. In 1959-60 we have recorded 5 cases.

Cases of cirrhosis liver are usually non-toxic and ambulatory. Their appetite is not grossly impaired and they are able to manage their routine duties in spite of ascites and oedema of the feet. But as liver cell failure gradually sets in, characteristic clinical changes occur. The earliest appreciable change is a blunting of alertness, and delayed reaction time, gradually progressing to drowsiness. The breath may be foul-smelling, the so-called fetor-hepaticus. This fetor may be absent in patients who have been on broad-spectrum antibiotics, or in those who have been on a low protein diet. But while its presence is very characteristic, the absence of fetor hepaticus does not eliminate hepatic coma. The coma gradually deepens. If jaundice had not been observed before the onset of coma, it becomes evident now, and often helps to clinch the etiology of the coma,—*viz.* liver cell

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failure. Jaundice however, is not intense in cases of cirrhosis liver while cases of acute yellow atrophy, infective hepatitis and toxaemias of pregnancy manifest well marked icterus.

As the coma sets in, careful observation may reveal an involuntary tremor of the outstretched hands, suggestive of the flapping of wings of birds. The occurrence of these tremors in a case of cirrhosis liver is of great import, and a sign of impending coma. In 50% of our cases these flapping tremors were observed in the precoma state. One case under our observation, however developed flapping tremors after the coma was well-established. In the early stage of coma, there is a vacant stare, when the patient appears awake, but does not answer questions, strongly reminding one of the coma vigil of typhoid. From this stage onwards the condition passes through a state of apparent sleep with lack of response to questions, to total lack of response even to nocuous stimuli, and finally to deep coma. Once coma sets in, one cannot be sure of the prognosis; death may occur during the the first attack, as early as within 24 hours, or as late as even after 10 days. In our 1958-59 series of 12 cases, 5 patients died after 1-10 days of coma, and 4 patients were taken home in a very bad condition, against medical advice. Only 2 cases out of 12 survived. The chances of survival depend on the precipitating factor.

Anaemia, confusion and hallucinations are common manifestations before the coma deepens. Patients may become noisy and unmanageable, and it may be possible to control them only under the delirium sheet and/or heavy doses of paraldehyde. Long acting narcotics with low toxicity are much preferable to short-acting drugs in these cases. Since the liver is the detoxicating organ, other types of narcotics especially pethedine, morphine and the short acting barbiturates like seconal sodium and soluble phenobarbitone, are contra-indicated. Paraldehyde in doses of 5-6 c.c. may be given slowly intravenously. It can also be given intramuscularly but action will be delayed.

The deep reflexes are grossly exaggerated and the plantar response is extensor on both sides. Nausea and stertorous breathing may develop as the coma deepens, and like as in all other comatous states, jaw-breathing may occur as a terminal manifestation. There is nothing characteristic of coma of hepatic origin except for icterus, which may appear in a small percentage of cases. The so-called flapping tremor either may not occur, or may occur after coma has set in, and it is seen in other conditions with, or without coma. When a man suffering from cirrhosis liver suddenly lapses into a state of unconsciousness, we may with confidence diagnose hepatic coma. The suddenness of onset varies from as short an interval as one to two hours to 24-48 hours.

Factors precipitating coma in a patient with a damaged liver, are fairly wide in number. Infection especially pneumonia, is responsible in a large number of cases. A bout of of haematemesis may precipitate coma; in this case, heavy loss of protein with rise in blood urea and fall in systemic pressure explain the failure of an already damaged liver, which cannot cope with the elevated non-protein nitrogen. Bleeding from sources like a gastric or duodenal ulcer may have equal disastrous results. Surgical operations—major or minor—and paracentesis abdominis often performed to relieve a patient's distress, have also been known to provoke coma in cases of cirrhosis liver. It cannot be sufficiently stressed that these patients tolerate hypnotics very badly especially the long-acting sedatives like phenobarbitone, pethidine or morphia. Diuretics like ammonium chloride, acetozolamide (Diamox), chlorothiazide and hydrochlorothiazide may aggravate a condition of liver damage to liver failure; chlorothiazide is less damaging than acetazolamide, and of the two, hydrochlorothiazide is peferable to chlorothiazide as the dosage is smaller and electrolyte balance less disturbed. Coma precipitated by these diuretics can be prevented by the administration of broad spectrum antibiotics like chlortetracycline or neomycin. Portocaval anastomosis and other shunt operations also provoke coma. These patients

tolerate dietary proteins poorly and in them the metabolic disaster may be checked or even prevented by maintining them on a low protein diet (15-20 mgs. per day for an adult); strangely this low protein intake does not aggravate the liver dysfunction nor induce hypoproteinaemia. In subjects prone to cholaemia, administration of methionine or methionine-like compounds favour the occurrence of coma. The derivatives of methionine like methyl-mercaptol or other sulphur containing substances may be responsible for the fetor hepaticus.

The dysfunction of the brain is attributed to the presence in the circulation of toxic substances probably derived from the intestines without undergoing the normal detoxication in the liver. The toxic agent may be from the food., *i.e.* protein products or from bacteria in the intestines. Extensive vascular derangement in the liver and the resulting portal hypertension are responsible for short-circuiting of the liver and transport of these toxic substances through collateral channels and the oesophageal plexus of veins, to the systemic circulation. The toxic material, may not therefore get detoxicated by the liver but pass from the portal vein through the hepatic to the systemic veins. The concentration of ammonia in the blood and C.S.F. has been found to be raised in hepatic coma. With a normal liver, the metabolism would have proceeded from ammonia formation to the formation and excretion of urea; in these cases the liver fails to form urea from ammonia. The ammonia theory however is not quite satisfactory because subjects of cirrhosis liver given large quantities of ammonium chloride (raising the blood ammonia value to twice or three times that achieved in hepatic coma) do not invariably develop coma. The blame is therefore shifted to the brain which is probably more sensitive and not able to tolerate this amount of ammonia. It may be more correct to talk of cholaemia—proneness rather than ammonia intoxication.

TREATMENT

Treatment aims mainly at elimination of the etiological or precipitating factors. Sedatives not tolerated by the liver, the use

of ammonium chloride and essential amino-acids like methionine in large doses should be discontinued.

If hematemesis occurs, it should be attended to immediately. Blood transfusion is urgently indicated in these cases. Dietary protein must be cut down to the barest minimum not exceeding 20-25 gms. for a time. The calorific needs of the body is maintained by giving essentially a carbohydrate diet particularly in the form of glucose intravenously. 40% glucose is advocated in these cases, but in this strength, glucose may cause thrombosis of the small veins. This may be avoided by the use of a polythene tube which is threaded through the cubital or femoral vein, and its free end allowed to reach one of the greater veins or superior or inferior vena cava. Thrombosis is not likely to take place when the polythene tube lies in one of the larger veins. One to two pints of 40% glucose may be given, with 20-40 units of crystalline insulin to fix it. Hypokalemia is common in hepatic coma as revealed by E.C.G. records; this may be combated by the administration of potassium. In patients with ascites sodium administration is not indicated; it does not improve the systemic pressure nor help it to eliminate the non-protein nitrogen; on the other hand, it may worsen the condition by causing water retention. If however the systemic pressure is very low, whatever its cause, the administration of noradrenaline in a slow I.V. drip will improve the pressure and cause a good diuresis. Steroid hormones have not given beneficial results especially in coma resulting from infective hepatitis.

Walsh advocated the use of glutamic acid in hepatic coma on the assumption that glutamic acid will react with ammonia to form innocuous glutamin. Our trials with glutamic acid in injectable form—*i.e.* glutamin I.V. have not resulted favourably, thus comparing equally with the experience of other workers in this field. However, once the patient recovers from the coma, glutamic acid renders him more alert and responsive. In this hospital, we have no experience with the amino-acid arginin.

Wide spectrum antibiotics are very useful in hepatic coma. It is believed that they clear away the normal intestinal organisms like b. coli, which occur in large numbers. Enemata and saline purges are also useful in this correction. These antibiotics are best administered parenterally. We usually advocate chlortetracycline 500 mg. in I.V. glucose once or twice a day. Neomycin and Kanamycin have not been tried due to non-availability of these antibiotics locally. Antibiotics are also useful in preventing coma in cirrhosis patients who are on medication with methionine or hydrochlorothiazide.

PROGNOSIS

The outcome of coma secondary to cirrhosis liver is always better than that which occurs after infective hepatitis. Prognosis depends upon the number and duration of each attack. The first attack may not be very severe but with each subsequent attack, the prognosis becomes steadily worse. In the last series, we have recorded a case which survived six attacks, but succumbed to the seventh. Generally only 3 or 4 attacks occur, death taking place in the 3rd or 4th attack. With each relapse, the delirium and coma become worse, longer lasting and less responsive to treatment. Institution of treatment early enough aids recovery considerably; coma lasting for over 7 days indicates a bad prognosis. One of our cases remained comatose for 14 days before he expired. The prognosis is said to be better in women than in men.

CONCLUSION

Records of hepatic coma are steadily increasing. Fair success in the management of cirrhosis is bringing more of these cases to view. The pathogenesis of the condition is still unsettled and we are yet in the stage of multiple theories. The accepted treatment is useful in early attacks, but the prognosis is far from

satisfactory. Prevention may be aided by discretion in the use of essential amino acids, withholding sedatives not tolerated by the liver, emergent treatment of haemorrhages, avoidance of alcohol and studied use of antibiotics.

I thank Dr. Sarada Menon for having gone through the article and given her valuable suggestions.

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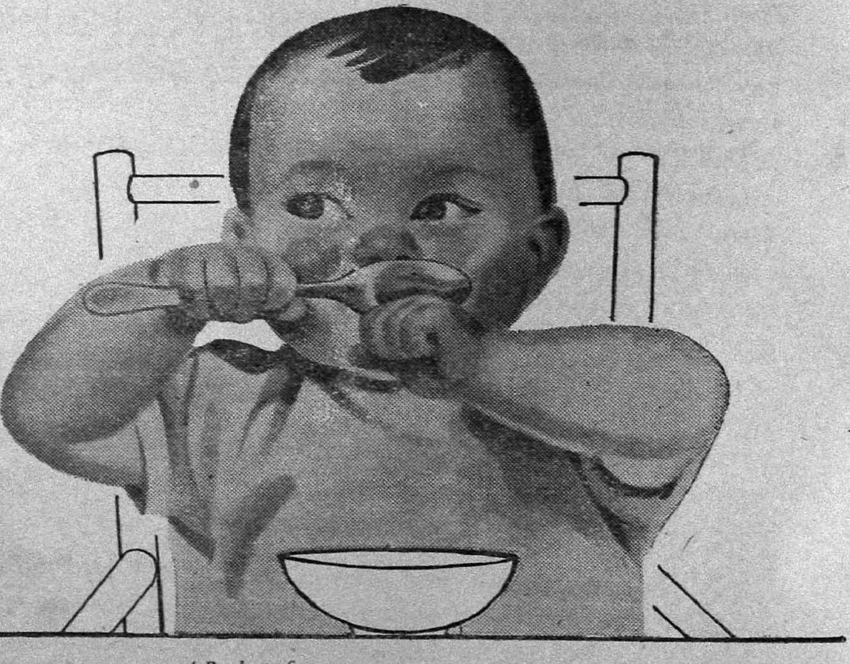

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We offer our sincere congratulations to Dr. T. Janardhanan, M.B.B.S., M.SC., Professor of Anatomy on his well deserved appointment of Director and Professor of the Institute of Anatomy, Stanley Medical College. After a very distinguished career beginning with the student phase and as member of the Madras Medical Service, clinical and non-clinical branches, he has distinguished himself as a capable teacher, a very capable administrator and a good friend of everyone of the medical profession. He has amassed a fund of knowledge in the discipline to which he belongs through his personal contact with the giants in the field, recently in the Western World and certainly in our opinion bids fair to become an invaluable gem in the Firmament of Directors.

A Study of the Pterion in South Indian Skulls

Dr. T. JANARDHANAN, M.B.B.S., M.SC.*
& Dr. T. JAYAVELU, M.B.B.S.†

INTRODUCTION

Pterion in man is the site of the anterolateral fontanelle and is formed by the junctional bones parietal, frontal, ali-sphenoid and temporal. A thorough analysis of the literature on the subject has been made by Ashley Montague (33). He quote authors like Burt and Turner, Ranke, Collins and Mescarenhas who worked about the later part of last century and early this century on 'Hindoos', 'Tamils' and 'Asiatics'. The materials woked on do not appear to be specific or sufficient except for Ashley Montague who examined 472 skulls of 'Asiatic Indians'. Again the materials are grossly generalised and refer to regional rather than racial groups. There are no comparable data available on this subject from the Indian Workers. Therefore, a re-study of the problem of pterion in South Indians has been taken up. The population of South India is an admixture of archaic-cacausoid (pre-dravidian) and cacausoid (Indo-dravidian), as the rest of India mostly (Montague, 51). A comparative study of the pterion of this group with the Australian aborigines, another archaic cacausoid group, is also outlined. Collateral study on a few skulls of Bombay and Bengal regions of India has also been included, though the number examined is not statistically significant.

MATERIALS AND METHODS

Materials examined were the collection available in the Madras Medical College, Madras. About 150 skulls were examined of which 10 each were from Bombay and Bengal.

*Professor of Anatomy, Director, Institute of Anatomy and Stanley Medical College, Madras.
†Asst. Professor of Anatomy, Stanley Medical College, Madras.

A total of 105 (92 South Indian, 5 Bengal and 8 Bombay) skulls only could be annexed for the study as the rest were found to be in advanced stenosis as to make the pattern of pterion not visible. There were 37 females (35 South Indian and 1 each for Bombay and Bengal and 68 males (57 South Indian, 7 Bombay and 4 Bengal). In cases where records of sex and age were not authentically available they were determined by the judgement of the authors. Line drawings of the pterion patterns given in figures 1 and 2 represent them as observed with the skull held in the position of Frankfort. Horizontal quantitative measurements were taken in relation to this plane.

OBSERVATIONS

The usual four type of pterion patterns *viz.*, sphenoparietal, fronto-temporal, epipteric and steliate were noted. A brief summary of the contacting bones is given below (Figs. 1 & 2).

Squamous Temporal bone.—This was found well developed and arched in its contact with the parietal and sphenoid, the height of it being in a level with or a little above the pteric junction. The anterosuperior angle was well marked, with a sharp or, rounded edge, in narrow sphenoparietal or stellate contacts with or without epipteric bones. The fronto-temporal contacts were of the "mediate" type with the process of the parieta as a quadrilateral expansion articulating with the other three bones. The suture of this process was either straight or curved and rounded.

Parietal bone.—Its anterior inferior angle which comes in relation with the pteric region was found dependent mostly on the height of ali-sphenoid. Contact with a low sphenoid or high narrow sphenoid was established by a process with or without epipteric bones. In stellate contacts this process was found to be sharp.

Frontal bone.—The posterior inferior part of the frontal comes in association with the pterion. The margins were found bevelled at the expense of the temporal surface in its contact

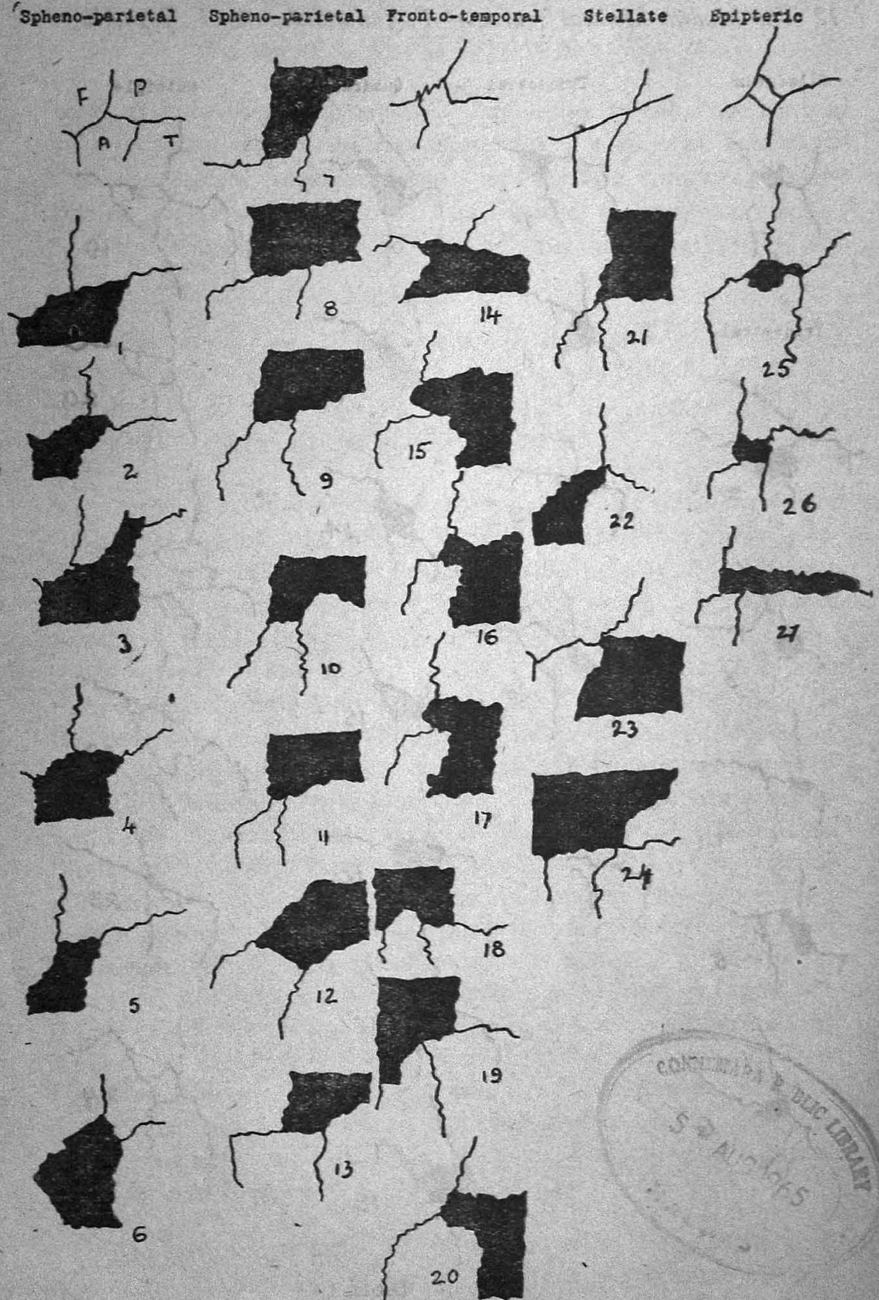

Fig No. 1

Examples of the types of Pterion contact and variations in the Pteric Bones. (After Murphy)

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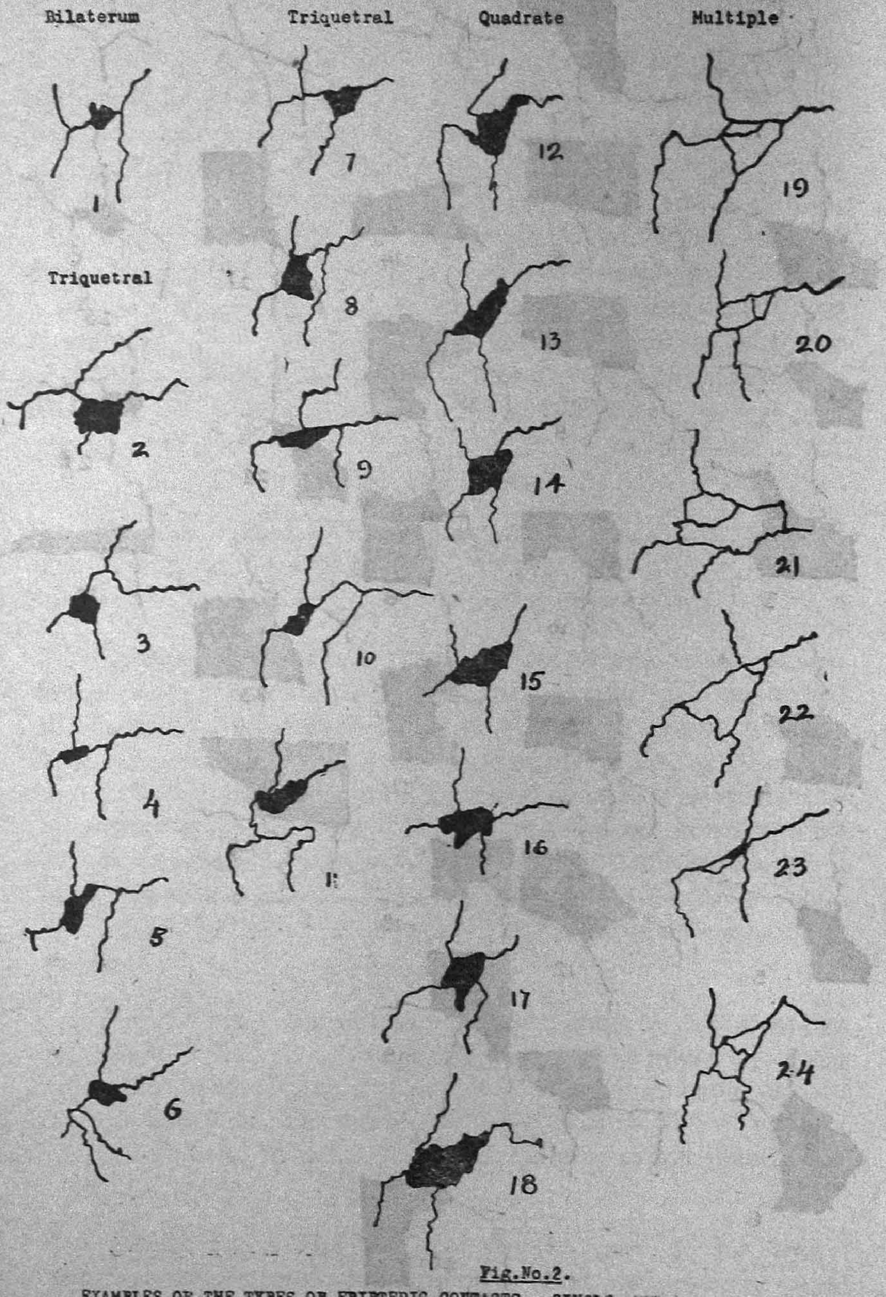


Fig.No.2.

EXAMPLES OF THE TYPES OF EPIPTERIC CONTACTS - SINGLE, MULTIPLE AND MIXED TYPES

(After Murphy)

with ali-sphenoid and parietal. The angle of the coronal and fronto-sphenoidal suture was about 130°. This angle was modified in case of fronto-temporal and stellate contacts. In the fronto-temporal contacts the contribution on temporal process of the frontal was found to be minimum except in one (Fig. 1-18).

Ali-spenoid bone.—This was broad or narrow, high or low. The pterion was dependant on the size of this bone. It was pointed in stellate; parietal prolongation, in case of high sphenoid, with or without epipteric bones.

Epipteric bones.—These are the small bones found ununited with the rest of the pteric complex. Altogether there were 47 sides with epipteric bones in South Indians; 14 bilateral, 17 with spheno-parietal and 2 with fronto-temporal contacts. Similarly for Bombay, it was 4 sides, 1 bilateral and 2 with spheno-parietal. For Bengal it was 4 sides, 1 Bilateral, 1 with Spheno-parietal and 1 with Stellate (see Table 1). The epipteric bones were of varied and irregular sizes and shapes. However they could be brought under two broad classifications as quadrilateral or triangular shaped bones establishing contact either with two pteric bones when it is called "os epiptericum bilaterum", or with three pteric bones called "os epiptericum triquetrum", or with all four pteric bones called "os epiptericum quadrilatrum". The multiple epipteric bones are referred to as "ossa epipterica". The classification is according to Montagu. The typing of epipteric bones according to Murphy is found not feasible since it does not admit of os epiptericum bilaterum and denies the existence of epipterics with frontal contact. This supposed absence is also confirmed by Bardeleben to advance the theory that epipteric is a post frontal bone. To this Montagu adds "this statement is somewhat obscure but is given here for what it is worth" (Montague '33).

The commonest type of epipteric bone was the quadrilateral type (type II and III Murphy). Next in order come the triquetral (type I Murphy), multiple epipterica (types IV and V

Table 1

Incidence of Pteric types in the South Indian, Bombay and Bengal

SKULLS

Type of Pterion Contact.		No. of Skulls.			Percent.			*Australian Aborigines.
Right.	Left.	S.I.	Bom.	Beng.	S.I.	Bom.	Beng.	
Symmetrical								
Sphenoparietal ...	Sphenoparietal ...	53	4	2	57·6	50	40	228-62%
Epipteric ...	Epipteric ...	14	1	1	15·2	12·5	20	31-8·4%
Frontotemporal ...	Frontotemporal ...	2	0	0	2·2	0	0	13-3·5%
Stellate ...	Stellate ...	0	0	0	0	0	0	0-0%
Asymmetrical								
Sphenoparietal ...	Epipteric ...	10	1	1	10·8	12·5	20	31-8·4%
Epipteric ...	Sphenoparietal ...	7	1	0	7·6	12·5	0	31-8·4%
Stellate ...	Sphenoparietal ...	2	1	0	2·2	12·5	0	1-0·3%
Sphenoparietal ...	Stellate ...	1	0	0	1·1	0	0	2-0·5%
Frontotemporal ...	Stellate ...	1	0	0	1·1	0	0	1-0·3%
Frontotemporal ...	Epipteric ...	1	0	0	1·1	0	0	5-1·4%
Epipteric ...	Frontotemporal ...	1	0	0	1·1	0	0	6-1·6%
Stellate ...	Epipteric ...	0	0	1	0	0	20	0-0%
Sphenoparietal ...	Frontotemporal ...	0	0	0	0	0	0	11-3%
Frontotemporal ...	Sphernoparietal ...	0	0	0	0	0	0	7-1·9%
Stellate ...	Frontotemporal ...	0	0	0	0	0	0	1-0·3%
Epipteric ...	Stellate ...	0	0	0	0	0	0	0-6%
	Total ...	92	8	5	100	100	100	368-100
Symmetrical ...	...	69	5	3	75	62·5	60	272-73·9
Asymmetrical ...	...	23	3	2	25	37·5	40	96-26·1

* From Murphy 1956.

Table 2

Percent of Pterion contacts treated independently of sides

Type of contact.	Number and percent.		
	South Indian.	Bombay.	Bengal.
Sphenoparietal ...	126 - 68·5%	11 - 68·8%	5 - 50%
Frontotemporal ...	7 - 3·8%	0 - 0%	0 - 0%
Stellate ...	4 - 2·2%	1 - 6·2%	1 - 10%
Epipteric ...	47 - 25·5%	4 - 25%	4 - 40%
Total ...	184 - 100%	16 - 100%	10 - 100%

Murphy) and bilateral (see Table 3). The fronto-parietalisphoid type of epipteric which was found to be non-existent in the Australian aborigines was seen in 4 out of 47 in South Indian, and in 2 out of 4 Bombay skulls. This type occurred only in high sphenoids that established contact with the parietal by a process (see Fig. 2 - 5 & 8). It was the parietal contribution of the pteric complex in high sphenoids that was replaced by this type of epipteric. But this was not invariably the order as both the parietal and sphenoidal processes coexisted together in high sphenoids without epipteric (see Fig. 1-3 & 7). The other form in which this was seen to occur as given in Fig. 2-4 & 9. This again suggests that it is the parietal process that is replaced.

Table 3

Type of Epipteric Bones and their distribution as to Sex and Side

TYPE OF EPIPTERIC BONE

Particulars.	Quadri-latum.	Triquetrium.				*Bilatraum.			Ossa epi.	Total
		FPA	PTA	FTA	FPT	AF	AP	AT		
South Indian...	22 4 fl 4 fr 7 ml 7 mr	4 1 fl 1 fr 2 ml 0	11 3 fl 2 fr 3 ml 4 mr	1 1 fl 0 0 0	2 1 fl 0 1 ml 0	0 0 0 0 0	1 0 0 0 1 mr	0 0 0 0 0	6 1fl 0 2 ml 3 mr	47
	46·8%	8·5%	23·4%	2·1%	4·3%	0%	2·1%	0%	12·8%	100%
		38·3%				2·1%				
Bombay ...	2 0 0 1 ml 1 mr	2 0 0 1 ml 1 mr	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	4
	50%	50%	0%	0%	0%	0%	0%	0%	0%	100%
Bengal ...	1 0 1 ml 0	0 0 0 0	3 0 2 ml 1 mr	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	4
	25%	0%	75%	0%	0%	0%	0%	0%	0%	100%

* Always in apposition with alisphenoid.

F - Frontal.
P - Parietal.
T - Temporal.
A - Alisphenoid.

fl - female left.
fr - female, right.
ml - male, left.
mr - male, right.

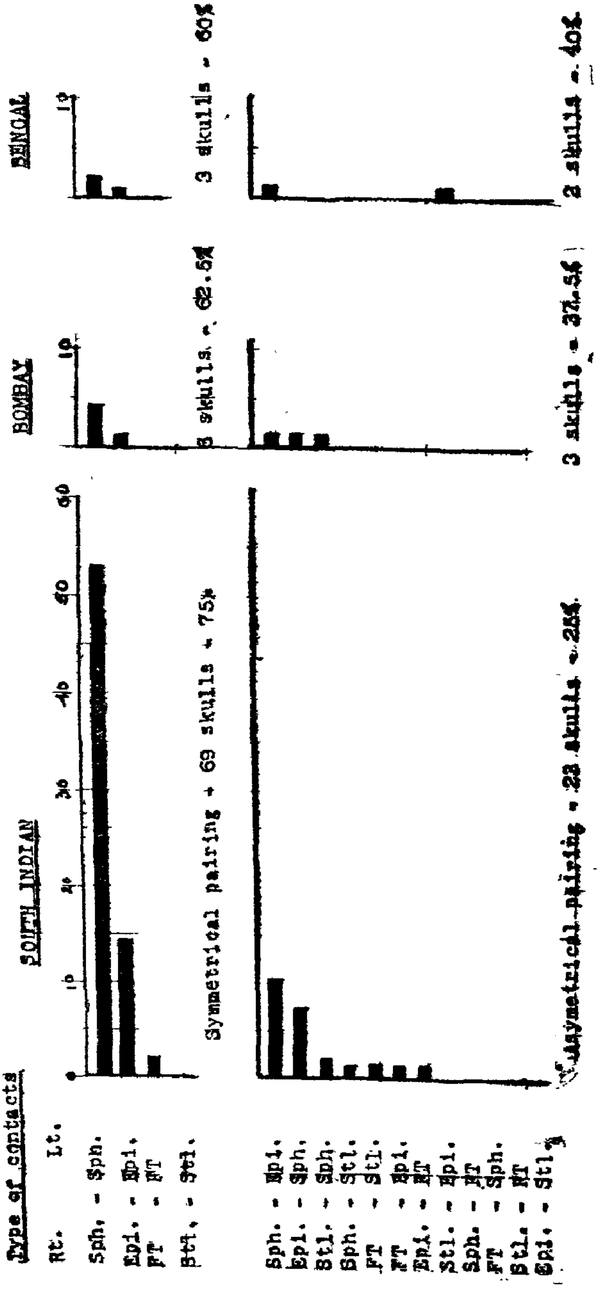


Fig. No. 3

Column Diagram on the incidence and pairing of types of Pterion contacts in South Indian, Bombay and Bengal Skulls.

There were 24 skulls with the skull cap removed and it was possible in them to observe the relation of the extension of orbito-sphenoid to the pteric region and epipteric bones. The extension of the orbito-sphenoid did not always follow the presence of epipteric bones; and the percentage as could be worked out from this study was (*i*) with contact but no epipteric (5 skulls), 20·8%; (*ii*) with contact with epipteric (3 skulls), 12·5%.

From the morphological observations made by Murphy (1956) the findings for the direction of the inter-epipteric sutures in the six skulls with multiple epipteric bones were: (*i*) Parieto-temporal 3; (*ii*) Parieto-frontal—2; (*iii*) Fronto-temporal—1 (See Fig. 2-19 to 24).

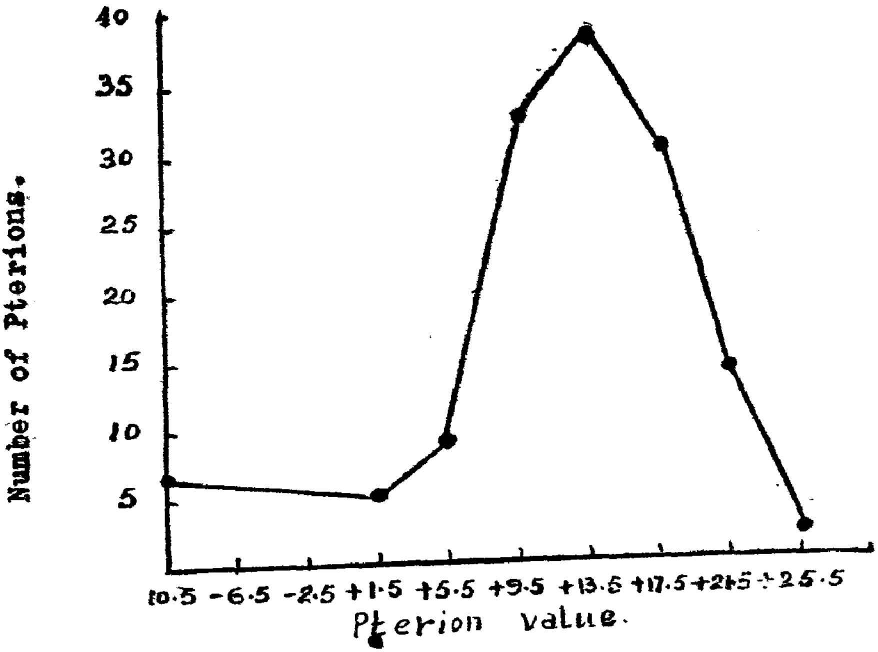


Fig. No. 4.

Frequency Distribution Curve of Pterion value to Number of Pterions in South Indian Skulls.

Mixed type of pteria.—(Fig. 2-1 to 11 and 19 to 24). This is possible only with triquetral, bilateral and ossa epipterics. In the last mentioned type the pteric bones were never seen to be involved directly. The figures in Murphy's work (1956) also confirm this finding. It is not known what morphological significance this bears.

The commonest form of triquetral association was with sphenoparietal contact. There were only three triquetrals with fronto-temporal contact (2-3, 6 & 11).

The majority of triquetral epipterics were found to be contiguous with parietal-temporal-alisphenoid. Its contiguity with fronto-parietal alisphenoid, fronto-parieto-temporal and fronto-temporal-alisphenoid were only next in frequency (see Table 3).

There was only one skull with os bilaterum between parietal and alisphenoid (see Fig. 2-1 & Table 3).

The triquetral epipteric in company with well developed frontal process of the temporal is significant in that it suggests that these two are morphologically variant. Normally the fronto-temporal suture is serrated but when the frontal or temporal process is ill-developed it is simple.

Incidence and pairing.—The usual 16 types of pteria are formulated, 4 bilaterally symmetrical and 12 bilaterally asymmetrical. The results obtained are found in table 1. It is found that bilateral sphenoparietal contact is the mode. Where this fails the union is by epipteric or fronto-temporal or stellate in that sequence.

In the asymmetrical series again spheno-parietal contact predominates with epipteric, stellate and fronto-temporal following in descending order. Distribution as to side is not significant in any of the series (see Table 1).

These observation held good for the skull of Bombay and Bengal as well.

Effect of sex and age.—Data for sex and age are given separately in Table 5. Age incidence has been grouped as pre-ossific and post-ossific periods, the determining factor being the union of basisphenoid and basi-occiput. This is deliberately chosen since the problem under investigation is one of growth and not a mere incidence in time. The inference from the table is that age and sex have no influence on the pteric pattern.

Table 4

Frequency Distribution of length of pterion in South Indian Skulls

(Pterion contacts with Epipteric Bones Excluded)

Class Interval (Millimetre)	Mid point	No. of Pterion	Percentage
— 12 to — 9	— 10·5	7	5·1
— 8 to — 5	— 6·5	0	0
— 4 to — 1	— 2·5	0	0
— 0 to + 3	+ 1·5	5	3·6
+ 4 to + 7	+ 5·5	9	6·6
+ 8 to + 11	+ 9·5	32	23·4
+ 12 to + 15	+ 13·5	38	27·7
+ 16 to + 19	+ 17·5	30	21·9
+ 20 to + 23	+ 21·5	14	10·2
+ 24 to + 27	+ 25·5	2	1·5
Total	...	137	100%

— Values refer to Fronto Temporal Pterion length.

O Values refer to Stellate Pterion length.

+ Values refer to Sphenoparietal Pterion length.

(Bombay and Bengal Skulls excluded from this investigation).

Table 5

Pairing of Pterion Contacts by Sex and Age in the South Indian, Bombay and Bengal Skulls.

Types of Pairing of Pterion	SEX INCIDENCE						AGE INCIDENCE					
	South Indian		Bombay		Bengal		South Indian		Bombay		Bengal	
	Male	Female	Male	Female	Male	Female	Pre-ossific	Post-ossific	Pre-ossific	Post-ossific	Pre-ossific	Post-ossific
SYMMETRICAL												
Sph. — Sph. ...	34 (59·7%)	19 (54·3%)	3 (42·8%)	1 (100%)	1 (25%)	1 (100%)	7 (77·8)	46 (55·5%)	...	4 (50%)	...	2 (40%)
Epi. — Epi. ...	9 (15·8%)	5 (14·3%)	1 (14·3%)	...	1 (25%)	...	1 (11·1%)	12 (14·5%)	...	1 (12·5%)	...	1 (20%)
Ft. — Ft. ...	...	2 (5·7%)	...	...	...	...	...	2 (2·4%)	...	...	...	...
Stl. — Stl. ...	...	...	...	...	...	...	...	...	...	...	...	...
ASYMMETRICAL												
Sph. — Epi. ...	6 (10·5%)	4 (11·3%)	1 (14·3%)	...	...	...	...	9 (10·8%)	...	1 (12·5%)	...	1 (20%)
Epi. — Sph. ...	5 (8·8%)	2 (5·7%)	1 (14·3%)	...	1 (25%)	...	...	9 (10·8%)	...	1 (12·5%)	...	...
Stl. — Sph. ...	2 (3·5%)	...	1 (14·3%)	...	...	...	...	2 (2·4%)	...	1 (12·5%)	...	...
Sph. — Stl. ...	1 (1·7%)	...	...	...	...	...	...	1 (1·2%)	...	...	...	...
Ft. — Stl. ...	...	1 (2·9%)	...	...	...	...	...	1 (1·2%)	...	...	...	...
Ft. — Epi. ...	...	1 (2·9%)	...	...	...	...	1 (11·1%)	...	...	...	...	...
Epi. — Ft. ...	...	1 (2·9%)	...	...	...	...	...	1 (1·2%)	...	...	...	1 (20%)
Stl. — Epi. ...	...	...	...	...	1 (25%)	...	...	...	...	...	...	...
Sph. — Ft. ...	...	...	...	...	...	...	...	...	...	...	...	...
Ft. — Sph. ...	...	...	...	...	...	...	...	...	...	...	...	...
Stl. — Ft. ...	...	...	...	...	...	...	...	...	...	...	...	...
Epi. — Stl. ...	...	...	...	...	...	...	...	...	...	...	...	...
Total ...	57 (100%)	35 (100%)	7 (100%)	1 (100%)	4 (100%)	1 (100%)	9 (100%)	83 (100%)	0 (0%)	8 (100%)	0 (0%)	5 (100%)

Quantitative studies.—As done by Murphy (56) the "pterion values" were obtained for skulls with sphenoparietal and frontotemporal contacts irrespective of sides (see Table 5). The epipterics were excluded from this investigation. A graph plotted with sphenoparietal values as positive, frontotemporals as negative and stellate as 0, shows a curve of error that has not the regularity of a normal curve (Fig. 4). The distribution of values around the central tendency is somewhat over a wide range and the curve is skewed to the left. There is a tendency to dip low at zero confirming that stellate contacts are few to find. The explanation for this could be that (*i*) the material examined is inadequate in quantity or (*ii*) that there is a factor(s) opposed to this type of contact.

Other correlative studies.—Include stenocrotaphy and cephalic index. The data obtained were in accordance with the method followed by Murphy (56) and this was done to allow for comparison of data with this work. Table 6 gives the values obtained. Material examined does not appear to be homogeneous. Allowing for genetic variation in this mixed population there does not appear to be any relation between cephalic index or stenocrotaphy and pterion.

Montague (33) says, "It would appear that the key to the study of evolution of the pterion region is afforded by the structure of the malar bone, the latter being understood to be the post-orbital or temporal flange of the zygomatic". With this he goes on to say that "the invagination of the malar post-orbitally first takes place in the semnopitheques and thereafter occurs uniformally in all higher primates". He further asserts that "one bone in the pteric complex invariably compensates for the reduction in another, and the type of compensation is generally constant and characteristic". On this hypothesis it was assumed that the human zygomatic bone still holds a phylogenetic significance on the pterion. With this in view the most horizontal distance between the centre of pterion half of pterion value and the centre of frontzygomatic suture at the

Table 6

Correlation of Bilateral Pterion with Cephalic Index and Post-Orbital with in South Indian, Bombay and Bengal Skulls

Type of Symmetrical Pterion	CEPHALIC INDEX						POST ORBITAL WIDTH (Millimetres)					
	South Indian		Bombay		Bengal		South Indian		Bombay		Bengal	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Spheno-parietal: Narrow ...	71·8	3·4	73·8	...	...	...	84·8	4·1	88·5	...	...	...
Spheno-parietal: Medium ...	76·8	5·4	74·7	...	74·2	...	87·2	3·1	92	...	89	...
Spheno-parietal: Broad ...	75·3	6·5	...	...	...	...	87·6	6·9	...	...	...	...
Fronto-temporal ...	81·2	...	...	...	...	...	82·5	...	...	...	...	...
Epipteric Bone(s) ...	75·4	3·7	76·3	...	73·4	...	86·4	3·6	92	...	87	...
Stellate ...	...	...	...	...	...	...	...	...	...	...	...	...

outer aspect of frontal and zygomatic processes, was measured with the skull held in the F.H. plane. Data collected was irrespective of side. It was assumed that the distance called the pterion distance is proportional to the orbital flange of the zygomatic bone. The aim of this investigation was to correlate this distance with pterion value to find out the relation, if there be, in the various types of pterion contacts.

The data collected for the study was confined only to spheno-parietal and stellate contacts of South Indian skulls because of the comparative largeness of data of the former and because of the 0 value of pterion in the latter which otherwise could not be independently analysed. Frontotemporal contacts were excluded from this study for want of sufficient material. Epipterics were also excluded for the obvious difficulty of locating a convenient bony point of reference.

The data for the frequency of occurrence of the two variables *viz.*, pterion value and pterion distance, is given in Table 7, A steriogram is erected to get a visual idea of the data

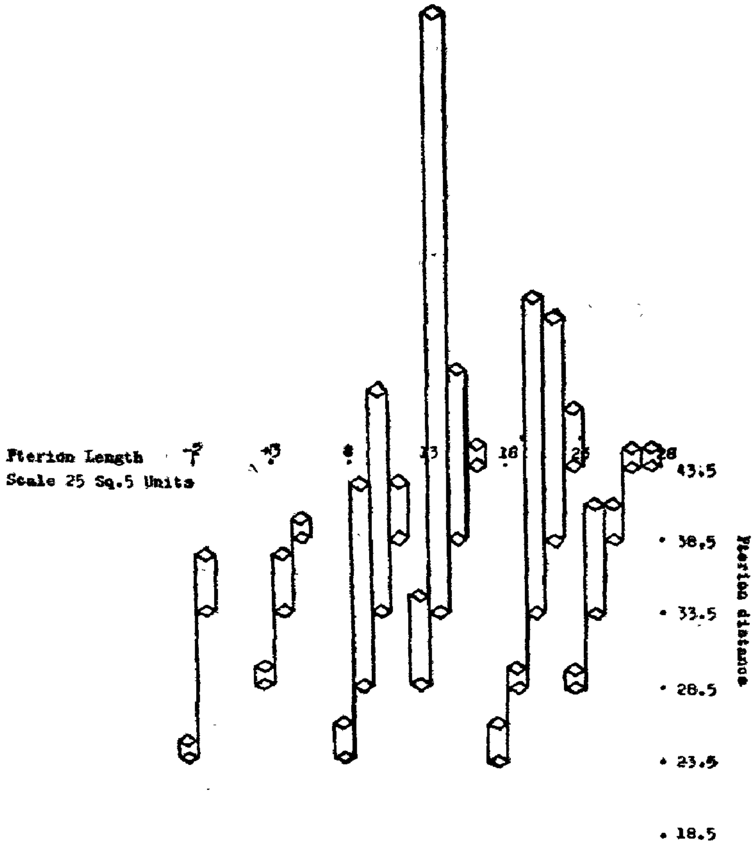
Table 7

Correlation Table—Pterion Length and Pterion Distance in Stellate and Spheno Parietal Pterions of South Indian Skulls

	Class interval (Millimetres)	Midpoint	PTERION DISTANCES					Total
			21 to 25	26 to 30	31 to 35	36 to 40	41 to 45	
PTERION LENGTH			23·5	28·5	33·5	38·5	43·5	
	— 4 to 0	— 2	1	...	3	...	...	4
	1 to 5	+ 3	...	1	3	1	...	5
	6 to 10	8	2	11	12	3	...	28
	11 to 15	13	...	5	32	9	1	47
	16 to 20	18	2	1	17	12	3	35
	21 to 25	23	...	1	6	2	1	10
	26 to 30	28	...	...	...	...	1	1
		Total ...	5	19	73	27	6	130

(Fronto-temporal and epipteric contacts excluded).
(Bombay and Bengal skulls excluded).

(Fig.5) Sections of the steriogram on the lines of regression give normal curves which show that the correlation surface within limits is normal. Based on this the inference could be that there is a relation between pterion value and pterion distance. The maximum frequency is between an average pterion value of 13·8 mm. with standard deviation 4·9 and average pterion distance of 33·4 mm. with standard deviation 4.



STEREOGRAM FOR PTERION LENGTH AND PTERION DISTANCE.

Fig. No. 5

Note.—Pterion distance and Pterion length shown as the axis form a horizontal plane on which third dimensional columns are erected. This figure is different from normal graph in that three variables are correlated as against two normally.

Eg., Against mid points - 2 and 18·5 there is no third variable; but against - 2 and 23·5 we get 5 units represented as 25 squares on a vertical plane.

For definitions of Pterion length and Pterion distance please see article.

DISCUSSION

The general characteristics of pteric bones in the present study do not appear to be different from Australian aborigines as given by Murphy (56).

Sphenoparietal contacts.—These predominate the rest of the types (see Table 2 and Fig. 3). Bilateral symmetry appears to be the rule (see Table 1). Of the 126 sides of sphenoparietal contacts in South Indians there were 35 narrow, 75 medium and 16 broad contacts. Similar figures for Bombay and Bengal were 1, 4 and 0 and 2, 9 and 0 respectively. Broad contacts were not found in latter group but this must be taken with reserve since the material examined was small in quantity. Comparative figures for sphenoparietal pterion from other authors as available from Montague (33) are: Mescarenhas 26 Hindoos, 84%; Montague 472 Asiatic Indians, 91·3%. These figures are definitely on the high side compared with the present finding (see Table 2). For Australian aborigines it is 539 sides for 368 Skulls (Murphy 56).

Frontotemporal contacts.—There are seven frontotemporal contacts on one or both sides in the present series for South Indians. No frontotemporal contacts were observed in the Bombay and Bengal skulls (see Table 2 and Fig. 3). Comparative figures for the other authors as obtained from Montagu (33) are given below :

Summated findings of Montague for various authors	Asiatic Indians	1273	2·3%
Montague	Asatic Indians	472	5·1%
Ranke	Tamil	27	7·4%
Collins	Asiatics	73	2·7%
Mescarenhas	Hindoos	26	7·6 (Bilateral 11.5% frontotemperal types).

The figures of Ranke and Mescarenhas are at variance with the present findings. In Australians it is 12% (Murphy 56). Figures for Europeans and anthropoids in this series as given by

Montague are: Europeans 26240 skulls, various authors, 1·9%; Gorilla 376, 98%, Chimpanzee 385, 81·8%; Orangutan 578, 29%; Symphalanges gibbon 28, 15·3% and Hyolobate gibbon 209, 19·4%. By a consideration of the frontotemporal contacts alone the Indian comes to be placed in an intermediate position between Australian aborigine and Europeans.

Stellate contacts.—There are four contacts of this type in this series in South Indians and one each in Bombay and Bengal skulls (see Table 2 and Fig. 3). Similar figures for other authors got from Montague (33) are: Mescarenhas 26 Hindoos 0; Montague 472 Asiatic Indians, 2 bilateral. Murphy observes 5 unilateral stellate contacts in a total of 736 sides of Australian aborigines skulls. Symmetry in stellates seems a rare phenomenon and asymmetrical types are found only in very low percentages. This is also confirmed in the graph in Fig. 2. The morphological significance of this is not known.

Epipteric contacts.—In the present study epipteric contacts of one or both sides come only next in order to sphenoparietal pterions (see Table 2 and Fig. 3). It is 25·5% for South Indians, 25% for Bombay and 40% for Bengal. Here also symmetrical pairing is greater than asymmetrical pairing and this has been noted in Gray's Anatomy (P. 328-46). The following are the findings for other authors Montague for this series.

Mescarenhas	26	Hindoos	30·7%
Montague	472	Asiatic Indians	5·9%

The low figures of Montague are not understood. Summated findings from Montague for epipteric contacts in Europeans and anthropoids are as follows:

Europeans	...	3451	12·7%
Gorilla	...	285	0·7%
Chimpanzee	...	240	0·4%
Orang-utan	...	146	Nil
Gibbon	...	83	1·2%

For Australian aborigines it is given by Murphy as 28·3%. The Indians and Australian aborigines come to be on a level high above the Europeans in this finding.

There are three unilateral pterions in South Indians Fronto-temporal contact 2nd epipteric bone being present together (see Fig. 2-3, 6 and 11). A survey of the literature does not reveal any finding of stellate contact with epipteric bone being present together. This is significant perhaps morphologically.

Table 3 gives the distribution of epipterics according to type, sex and side. There is no evident correlation amongst these.

Correlative studies.—It is said that cranial index, postorbital width (so called stenocrotaphy), presence of spine of sphenoid, cranial flexion angle are some of the determining factors of pterion pattern. In the present study only the first two are correlated and they are found to bear no relation (see Table VI).

As for the relation of zygomatic bone aud pterion pattern, Table 7 and Figure 5 appear to concede some relation. Whether this is true or apparent needs to be probed into by further statistical analysis with greater data.

CONCLUSION

In the pterions sphenoparietal contact is the norm and fronto-temporal the aberrant. The theory of frontotemporal contact being established through epipteric fusion (Cunningham 51) cannot be entertained because of the presence of both epipteric bone and frontal-temporal process in the pterion site (Fig. 2-3, 6 and 11).

An examination of Table 3 will bring out the orderliness of occurrence of epipteric bones on the pteric site, perhaps involving some genetic or mechanical factor. This rules out the

possibility of epipteric bones being equated with wormian bones (Keith '48).

Medwick and Washburn ('56) say *that in a thin skulled*, large brained animal like a man "the relative growth periods of the various vault sutures should determine the final form to a great extent than in any other animal. The thinness of the human vault bone shows that oppositional and resorptive processes are reduced to a minimum. This suggests that the study of the sutures and not bones or whole skulls will be the most useful way of analysing the variation in human skull form". Perhaps here lies the solution to the study of the pterion which, as suggested by Murphy ('56), has not yet attained a phylogenetic stability.

SUMMARY

1. 105 skulls of South Indian, Bombay and Bengal regions of India were examined and a record presented.

2. Sphenoparietal contact is the common type with epipteric, fronto-temporal and stellate following in succession. Side, age, sex, cranial index or post-orbital width have no bearing on the form of pterion.

3. Epipteric bones are classified according to shape and site of occurrence. The orderliness of occurrence of these bones suggests that they are different from wormian bones. There were four fronto-temporal contacts with frontal process and epipteric bone present together. Triangular epipteric contiguous with frontal-temporal-alisphenoid bones was found in four South Indian and two Bombay skulls. The method of occurrence is described.

5. An attempt has been made to establish a relation between zygomatic bone and pterion. More statistical work is suggested to be done.

6. The significance of pterion in ethnic grouping is focussed in places.

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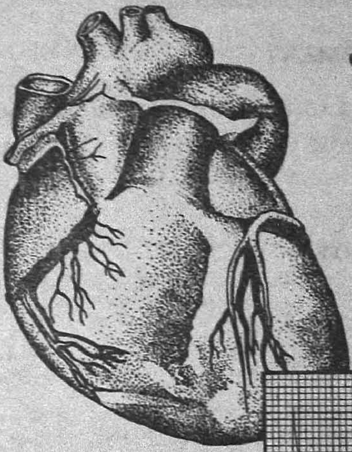
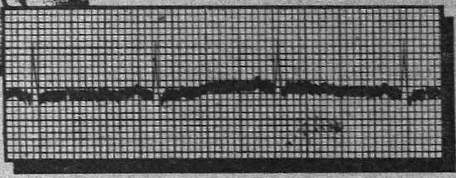


• Myocardial Damage
• Cardiac Insufficiency.



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Syphilis and Infancy

A RESUME

Dr. P. N. RANGIAH, M.D.*

In the first of the series of the Elizabeth Mathai Endowment Fund lectures delivered under the auspices of the University of Madras, first generation syphilis, in which the female sex partner was found to be involved concurrently both in the physiological event of pregnancy and the pathological episode of syphilis, was touched under the caption 'Syphilis and Pregnancy'. Trans-placental spread of syphilis from the untreated pregnant woman to the fertilised ovum in her womb was described and the remark was made with relevant supportive data of the pathologists, "How strange it is that the treponema pallidum invades the developing human zygote of 16 weeks or older but none younger than this fixed foetal age!".

Much emphasis next was laid on the disastrous outcome of pregnancy when syphilitic pregnant women were left untreated for syphilis for either want of facilities or through sheer complacence and/or ignorance. The relative merits of the outcome of treatment of syphilis in pregnant women compared with what obtains in a control group of pregnancies in non-syphilitic healthy women were pointed out at the same time.

While on this subject, a reference was also made to a particular such study in U.S.A. to pinpoint the following:—

A. That among the untreated syphilitic pregnant women, births of live, non-syphilitic infants approximated to 18% only; while (a) *foetal deaths*, (b) *prematurities* or (c) *the number of live born that scarcely survived the rigors of early life and died in the neo-natal*

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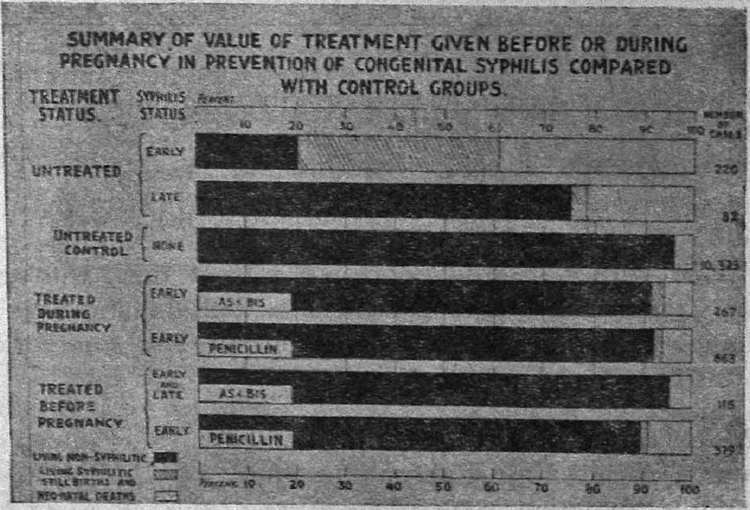
period, besides (d) *a proportionately* large crop of unfortunate infants that developed manifest syphilitic lesions subsequently, at various age periods, comprised the remaining 82%.

B. That on the contrary, if the pregnant syphilitic women were given the appropriate timely and adequate treatment the outcome of pregnancies very nearly equalled, if not even excelled, the happy results obtainable in the control group, *vide* Chart 1.

Chart 1



Thus much importance was focussed on the preventive aspect of syphilis in women prior to or during the period of pregnancy to avert the high foetal mortality and morbidity in the large segment of unborn innocent offspring.

Looking out elsewhere into the related affairs of another foreign country in the world it was expressed that the truism, "No syphilitic mother, no syphilitic baby", must have been the keynote to the Scandinavian success in the eradication of prenatal syphilis there. Dr. Weleander will be long remembered for the monumental homes in Scandinavia named in his honour and specifically erected for the reception, examination and all

round management of the syphilitic live born offspring. The speaker recalled his visit to a Weleander Home in 1952. While he was then most anxious to study the methods of management of congenital syphilitic babies there, he was most agreeably disappointed to find none such as this medical commodity had by then ceased to exist in that country. He had learnt also that the birth of a congenital syphilitic baby is considered an unholy reflection on the activities of the Public Health Service. Certainly it must therefore be a matter for great pride to the people of that country that first generation syphilis in women has been so successfully controlled through the enlightenment of the common man and the application of stringent public health measures inclusive of propaganda and prophylaxis. In short, it was reiterated that syphilis in infancy or congenital syphilis or in the present context, second generation syphilis had become extinct in Scandinavia.

Whilst this was so elsewhere, the pertinent question posed was, "What is the situation in Madras?". In answer to this, a reference was made, once again, to the figures furnished by the Pathologist of the Madras Corporation and the Superintendent of the Government Hospital for Women and Children, Egmore, Madras - *vide* Statistics, Chart 2.

Chart 2

Number of syphilitic children from syphilitic mothers calculated for a five year period

Clinics	Total No. of Females	Mothers with positive reaction	Living syphilitic infants	Still Births	Miscarriages
Corporation Antenatal Clinics ...	66533	3792	1550	690	257
Government Hospital for Women and Children, Madras ...	51695	4301	1760	782	292
Total ...	118228	8093	3310	1472	549

It will be gleaned from the data that in between these several institutions, there were discovered through serologic screening of 118,228 ante-natal cases over a period of 5 years, beginning 1953 and ending 1957, 8093 sero positive reactors amongst the parturient clientele.

Stemming from this factual statement is the necessary corollary, " What would have been the outcome of pregnancies among the 8093 parturient positive reactors ? ". At best the answer may be divided into two parts :

Firstly, conceding all the positive reactors among the pregnant women *to be early luetics* and that every positive sero-reactor in pregnancy had been given appropriate anti-syphilitic treatment, the results would equal, if not even excel those good results accruing in the non-syphilitic control group as per the representation in Chart 1. But the chances are that most of these parturient and positive sero reactors could not have had the standard treatment ; in which case the second possibility, calculated as per outcome of the control group of *untreated syphilis in pregnancy*, may be conjectured in terms of a bumper crop of foetal deaths, prematurities and births of living syphilitic infants ; mathematically calculated, the Statistician presented the appalling figures : a composite total of 2000 for foetal deaths and prematurities and 3310 living syphilitic infants.

Discussing foetal deaths, the Expert Committee of Health Statistics of W. H. O. pointed out that ' a foetal death shall be defined as death of a foetus prior to complete expulsion or extraction from its mother, irrespective of the duration of pregnancy and the death is indicated by the fact that after such a separation, the foetus does not breathe or show any other evidence of life such as the beating of the heart, pulsation of umbilical cord or twitching or movement of the voluntary muscles '.

Foetal deaths, according to same authorities, may be classified under three groups :

First Group.—Less than 20 complete weeks of gestation.

Second Group.—Between 20 and 28 weeks, and

Third Group.—28 weeks or more.

The adoption of the term "foetal death" referred to here is likely to be more useful of application and universal usage, since the other familiar equivalents such as 'abortion' and 'miscarriage' mean differently for different people.

To what an extent is syphilis responsible for foetal deaths? The pertinent available information is recorded hereunder: (1) Autopsied 164 dead born; of them 3% were due to syphilis (Ratnavathy and Reddy), (2) Arey and Dent have reported on autopsy findings over infants dying in the neo-natal period and they recorded that among 137 dead, congenital syphilis was discovered in 9, yielding a percentage of 6, (3) Gault and Jayaraj of Vellore reported on an autopsy done on a female baby that took only one breath and died. The body weighed 1500 grammes and presented a swollen abdomen. Among other details mentioned it is noted with interest that numerous spirochaetes were seen in sections of intestines, liver, spleen and pancreas and the liver sections revealed the presence of fibrous tissue in the region of the portal tract *and in between the liver cells.* There is thus available sufficient proof that syphilis also plays an etiological role in the causation of foetal deaths.

What constitutes prematurity? The definition of this term also requires elucidation. As noted in the Manual of International Statistical Calculation on Diseases, Injuries and Causes of Death, a premature infant is a live born infant with a birth weight of 5-1/2 lbs. or 2500 gms. or less. In certain countries, the weight is not specified. A live born infant of less than 37 weeks of gestation is premature.

What is the significance of prematurity? Premature births are more common amongst untreated syphilitic pregnant women than amongst non-syphilitic women. It is about 11 times the

figure for non-syphilitic group and Cruickshank has pointed out that syphilis is an important cause of premature delivery and prematurely born infants are much sooner carried away by bronchopneumonia, asphyxia or atelectasis. There were prematurities recorded in the annual report of the Health Officer of Corporation of Madras for every year.

The significant role of preventable syphilis in the production of foetal deaths and prematurities having been explained, it may be seen what an enormous responsibility of national importance has fallen on the Obstetricians to discover the positive sero reactors among the ante-natal clientele and to put a full stop with requisite therapy to the large toll of mortality and morbidity that otherwise will overtake the innocent unborn in our area.

The subject of live born syphilitics was next taken up for consideration with particular reference to the period of infancy, It may be observed here at the outset how embarrassing it is that the term infant is difficult of comprehension, *e.g.* (i) a reference to Oxford Dictionary reveals that according to law a person under the age of 21 years is an infant! (ii) Another definition is, "an infant is the offspring, male or female of human parents from the earliest period of life upto 7 years of age". (iii) The medical dictionary indicates that the word infant is derived from Latin, infans, meaning speech and refers to the period of life upto 2 years. From this medley we may reasonably choose for our study the definition that an infant is a human being from the moment of expulsion from the maternal body upto 2 years of age.

For convenience and lest the speaker should trespass into the boundless subject of adult or acquired syphilis, care is taken to largely restrict the subject under review to Syphilis in Infancy.

It may be recalled that syphilis in infancy when contracted before birth is known severally as congenital syphilis, hereditary syphilis or pre-natal syphilis; or may also be spoken of as second

generation syphilis; and is an excellent example of "neglected syphilis", since out of negligence of first generation syphilis arises second generation syphilis.

The figures for "Congenital syphilis" in the statistical reports of the Institute of Venereology stand at 3% for the period of 10 years from 1948 to 1957 - *vide* Chart 3.

Chart 3

Years	Total Syphilis	No. of Congenital Syphilis	Males	Females	Percentage
1957	2353	49	21	28	2·90
1956	2636	72	30	42	3·03
1955	3537	91	34	57	2·80
1954	5326	116	72	44	2·10
1953	5642	114	55	59	2 50
1952	6867	129	63	66	2·40
1951	5806	126	56	70	3·03
1950	4772	124	57	67	3·10
1949	4973	133	53	80	3·50
1948	4888	76	33	43	3·70
		1030	474	556	
			46·02%	53·98%	

1030 cases of congenital syphilis were reviewed. Interesting high lights of the breakdown with comments are:

(*i*) The sex-war proportion is 474 males and 556 females or 46·02% and 53·98% respectively.

(*ii*) In the midst of the obstetric disasters overtaking syphilitic pregnant women, it is curious observation that Nature manages to send a relatively larger proportion of female congenital syphilitics alive into the world. The numerical preponderance of female syphilitic infants over male syphilitic ones is obvious. An infant brought for demonstration of congenital syphilis was also a female.

(*iii*) The study of the records over the 10 year period (*vide* Chart 4) from the standpoint of the rank of the infected babies born, reveals that the percentages of congenital syphilitics

belonging to the first, second, third and fourth para went down in the descending order, proportionately, finally dwindling down to insignificant figures, confirming the belief that congenital syphilis is more common among the first few pregnancies in a woman's obstetric career in our study.

Chart 4

Rank of the congenital syphilitic infant

Year	Total No. of Cases	1st Para	2nd Para	3rd Para	4th Para	5th Para	6th Para	7th Para	8th Para	9th Para	10th and above	Un-known
1957	49	6	10	4	6	3	4	4	1	...	...	11
1956	72	11	6	6	4	6	2	3	1	1	...	32
1955	91	7	7	10	6	6	5	1	3	2	...	44
1954	116	20	20	11	18	4	4	3	2	...	1	33
1953	114	10	22	15	19	5	5	2	2	1	1	32
1952	129	21	30	19	15	15	10	1	1	...	1	16
1951	126	32	12	21	12	12	6	4	2	2	2	21
1950	124	16	26	28	11	10	10	2	2	2	...	17
1949	133	21	29	16	14	10	8	3	2	5	1	24
1948	76	14	18	15	6	3	7	2	1	...	...	10
Total	1030	158 16%	180 17%	145 14%	111 11%	74 7%	61 6%	25 2%	17 2%	13 1%	6 1%	240 23%

(*iv*) With reference to the age of the infant at the time of its first exodus from the composite province of the obstetrician and the pediatrician to the Institute of Venereology, it will be seen that the large majority of infants happened to be within the first 6 months of age *vide* Chart 5. This aspect of the presentation of the neonates in the earliest period of life in large numbers has to be borne in mind in the calculation of failure rate, for it will be seen that in spite of the best of treatment afforded to them during this age period, many of the treated patients succumbed; whether this be due to the overwhelming luetic infection itself or because of unmanageable concurrent other infections or treatment reactions it is difficult to decide. Observers in other lands have recorded similar disappointing outcome of the management of neglected syphilitic cases during the first 6 months of the age period of infants.

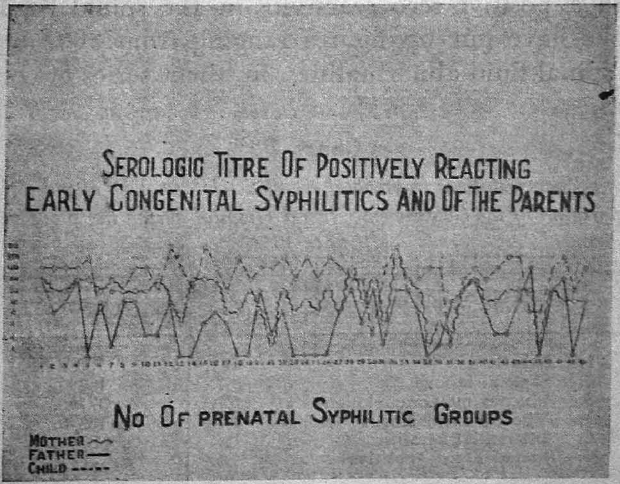
Chart 5

The age period when the congenital syphilitic infant was brought for treatment

Year	Total No. of Cases	At Birth	3 mths.	6 mths.	9 mths.	1 year	1½ years	2 years	3 years	4 years	5 yrs. and above	Un-known
1957	49	5	17	11	1	2	4	2	1	1	5	...
1956	72	13	27	13	6	3	2	...	...	...	8	...
1955	91	6	25	14	8	8	3	...	...	4	18	5
1954	116	9	21	19	16	12	6	1	3	...	26	3
1953	114	4	36	23	13	9	2	2	...	3	18	4
1952	129	19	43	32	7	4	3	...	2	...	16	3
1951	126	8	49	28	9	8	5	4	2	...	9	4
1950	124	13	39	26	10	10	9	2	...	...	10	5
1949	133	8	50	24	19	16	1	...	1	...	11	3
1948	76	5	27	17	13	7	4	2	...	...	1	...
Total	1030	90 9%	334 32%	207 20%	102 10%	79 7%	39 4%	13 1%	9 1%	8 1%	122 12%	27 3%

(*v*) The serology of the congenital syphilitic infant was invariably higher in dils. than that of the mother while the father's was low or nil reactive, *vide* Chart 6.

Chart 6



(*vi*) At this juncture, it may be mentioned that the syphilitic new born may arrive into the world with no obvious external manifestation and the apparently normal prenatal

syphilitic infant may exhibit signs of syphilis within a few weeks after birth. The manifest lesions of early prenatal syphilis confined to the first 2 years of age of the prenatal syphilitic may be cutaneous, skeletal, muco-cutaneous and or visceral. Chancres are conspicuous by their absence. Special organs and the central nervous system among the systems may show outward evidence of involvement. The cardiovascular system maintains strict neutrality.

(*vii*) According to our statistics - for the period 1948-57 *vide* Chart 7, amongst the 1030 cases, the number of patients with skin lesions heading the list was 733. Next in order was the skeletal group standing at 467. Third in rank was the group of muco-cutaneous lesions putting up 277. Visceral involvement was observed in 133. Central nervous system was involved only in 6 cases of whom were 3 patients - 2 months, 3 months and 5 months old (Ref. VDOP 119/50 female, VDOP 92/50 female, VDOP 655/50 female) respectively. All these were blood sero-positive reactors who exhibited an increased cell count and a positive sero reactivity in the spinal fluid. Jeans and Cooke have put up figures ranging from 30% to 40% as showing spinal fluid abnormalities in their series of congenital syphilitics.

Chart 7

Frequency of manifestations in prenatal syphilitic children who attended the V. D. Department of the Government General Hospital during the 10 years—1948 to 1957.

Manifestations		Frequency
1. Skin lesions	...	733 times
2. Skeletal lesions	...	467 ,,
3. Mucous membrane lesions	...	277 ,,
4. Visceral involvement	...	133 ,,
5. Interstitial keratitis	...	37 ,,
6. Spinal Fluid—Positive	...	6 ,,

*C. S. F. Positive Cards:
1. 1970/56
2. 10394/54
3. 8069/54
4. 92/50
5. 119/50
6. 655/50

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Syphilis and Infancy

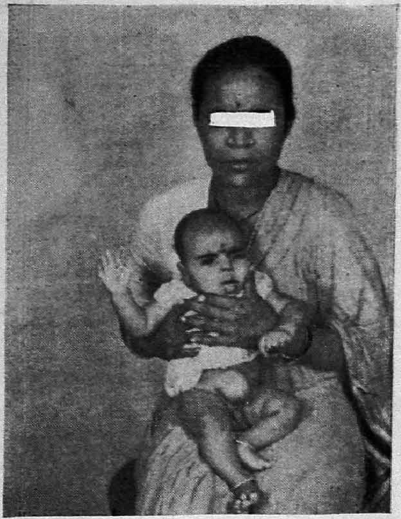

Fig. 1

The Story of the Mother and her Baby to bring out the Passive Transfer of the Maternal Reagin Across the Placenta into the Infant's Blood Stream

The picture presents the figure of a woman who came to us singly, but now carrying the baby, once in her womb. She was found to be a frank syphilitic with the dangerous potentiality of infecting her unborn offspring with the syphilitic organisms. She had a previous history of disastrous obstetric record. Her blood was reactive in high dilutions. There was not an easy method of determining if the foetus was already infected. However on the presumption that the chances of transplacental syphilitic infection are always present beyond the 16th week of intrauterine life, the woman's condition was taken for an emergency and was given the routine course of anti-syphilitic treatment with P. A. M. 2 c.c. or 6 lakh units I. M. daily for 10 days. As will be referred to in the subsequent presentation, the pregnant woman delivered at Full Term, an apparently normal non-syphilitic live baby. Examination of both the parent and the baby recently, yielded the only positive finding, in either of them, of a reactive serology. What is the significance of the positive serology in the infant? Does it connote syphilis? There could be any one of the possibilities regarding the positive serology.

(*a*) Technical false positive reaction.

(*b*) Biologic false positive reaction.

(*c*) True positive reaction.

(*d*) Treated residual reaction.

(*e*) Passive transfer of the reagin.

By a careful sift study it was resolved that the positive reactivity of the child belonged to the last category—passive transfer of the reagin. The baby was not treated, but was re-examined after an interval of a few months. The serologic titre declined almost to negativity and the infant was declared non-syphilitic.

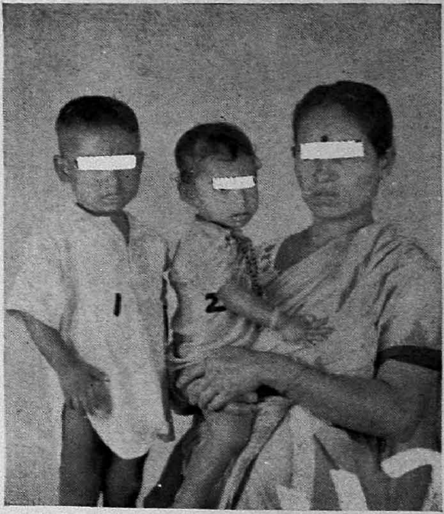

Fig. 2

The mother in the picture was first seen at the Institute in a state of pregnancy, when clinically she was suffering from an ano-proctitis with stricture of lymphogranulomatous origin. Her blood serology was reactive by the VDRL slide test in 1 dil. She was pregnant for the third time with a background of bad obstetrical antecedents in the past. Her husband (1387/57) on confrontation was clinically and serologically negative. Syphilis was not considered in the patient. She was treated with sulphonamides for the viral infection. She disappeared from our purview at the time of labour and reappeared later with an infant in her arms, who presented a high titred positive serology and typical roentgen lesions in the tubular bones. Both the mother and the child were treated with anti-syphilitic remedies.

Later, after a lapse of time the same woman appeared with one more baby in arms beside the first and another in her womb. She is still reactive by the VDRL slide test in one dilution. What is the destiny of the foetus? Does the mother require anti-syphilitic treatment as prophylaxis to protect the developing foetus?

As the confinement, immediately preceding the latest pregnancy, resulted in a normal baby seen in the picture as the younger sibling with no clinical, serological or radiological sign or stigma the mother is not given any anti-syphilitic treatment. It remains to be seen what the outcome of the latest pregnancy will be. The low titred positive serology in the mother is indeed a riddle.

(*viii*) Another category of prenatal syphilitic infants is the latent congenital syphilitic group. There were no figures for this entity. In the study of this group after eliminating technical false positive sero reactions and biological false positive sero reactions, care must be taken to weed out those cases with a passive transfer of syphilitic reagin as exemplified in the following case: a woman patient attended with pregnancy and a positive serology of 128 dilutions. A full course of anti-syphilitic treatment was given. Subsequently she was under surveillance till and after her confinement. She brought forth a female child after full term. A review examination of the mother and the child revealed no evidence of clinical syphilis. The mother's serology remained about the same. The infant's blood was reported positive in 8 dilutions. No treatment was given because of the presumption that the positive serology in the infant could be due to the circulation of passively transferred maternal syphilitic reagin in the infant's blood stream. It turned out on further follow-up examinations that there was no clinical evidence of syphilis or a rise in the serologic titre of the infant, but that a decline in the serology had been registered. Here the infant was not syphilitic and the question of latent syphilis or the necessity for anti-syphilitic treatment did not arise.

(*ix*) True latency in adult acquired syphilis is indicated by a persistent positive sero-reactivity or a rise in titre in the absence of any active clinical manifestations on the surface of the body, in the special organs, the cardiovascular system or the central nervous system, confirmed by negative roentgenograms of the cardio-aortic apparatus and negative cerebro spinal fluid studies. This is equally true of congenital syphilis. There might however be stigmata of congenital syphilis associated with a positive serology. Such cases are not rare in our experience.

In order to depict the various overt clinical manifestations referred to, photographs corresponding to various lesions were projected on the screen. A few of them are particularised for discussion hereunder :

(*a*) Under the category of skin lesions a special reference was made to bullae occurring in the palms and soles of a congenital syphilitic, teeming with treponema pallida on darkfield illumination examination. Bullous syphilides are not however common in adult syphilis.

(*b*) Describing muco-cutaneous lesions, the picture of an infant's eyes, was referred to. The welling pus from the conjunctival sac was found to show treponema pallida. Ordinarily it might have been expected that the ocular discharge was gonococcal in nature. In this instance both gonococci and treponema pallida were found.

(*c*) i. Referring to slides demonstrating skeletal lesions it was emphasized that the ends of tubular bones were a favourite resort of the treponema pallida because of the marked vascularity, slow circulation and low temperature, obtaining in the metaphyses where the diaphyses end and the epiphyses begin. The cartilage of conjugation when affected manifests signs of chondro-epiphysitis. Diaphysitis and periostitis also occur. It was mentioned that in the course of travels abroad by the speaker, no opportunity was available to study congenital syphilitic skeletal lesions either in the U.K. or in the U.S.A., but at the Hospital for the Sick Children in Great Ormonde Street, London, autopsy notes were available; and the collection of bones on congenital syphilis, a precious contribution of Jules Parrot, were studied. Of the categories of bone lesions in congenital syphilis it was pointed out that chondro-epiphysitis was the commonest on clinical examination confirmed by the skiagraphic proof of metaphysitis in the corresponding zone.

ii. Generalised periostitis and osteomyelitis also occur. But localised bilateral osteomyelitis in the region of the medial and upper end of either tibia was pathognomonic of

congenital syphilis. While on this subject, a reference in passing, was made to the etiologic diagnosis of macerated foetuses. It is known that syphilis and Rh incompatibility, both produce macerated foetuses. When in a diagnostic predicament and the etiological issue has to be decided between congenital syphilis and Rh incompatibility, the decisive factor is the skiagraphic demonstration of the bilateral 'cat bite lesion' of the medial and upper end of either tibia named after "Wimberger".

iii. Under reference to skeletal lesions in congenital syphilis again, a skiagram was shown exhibiting dactylitis. In syphilis of the patient under study the proximal phalanx was involved resulting in the contraction of the finger in contrast to skeletal tuberculosis in another infant in whom the terminal phalanx was affected, terminating in sinuses.

iv. Commenting on the "hot cross-bun skull" with cranio tabes and Parrot nodes, once upon a time attributed to syphilis, it was pointed out that the consensus now is that the thinning of flat bones of the cranium and sponginess of the frontal and parietal bones with prominence are largely due to nutritional deficiencies causing a metabolic derangement. This was mentioned to alert the practitioners on the change of views regarding cranial bone lesions in prenatal syphilitics.

v. To complete the story of bone lesions in Congenital Syphilis, sabering of the tibiae and prominence of the inner end of a clavicle - Higoumenakie sign - in a tardive congenital syphilitic were demonstrated as also the contracture of the little finger in a patient - the *Dubois*' sign.

(*x*) Discussing visceral involvement in congenital syphilis, a reference was made to text books, wherein splenomegaly is mentioned as a common occurrence. In the experience of the speaker, contrarywise, hepatomegaly is more frequent in our cases with an occasional splenomegaly occurring concurrently. As far as hepatomegaly is concerned, such patients were occasionally brought to the Institute presenting exclusively a protuberant abdomen and concurrent overt jaundice. Impression smears of

liver biopsy specimens obtained from living congenital syphilitic patients revealed treponema pallida on dark-field illumination examination. This demonstration of t.p. in biopsied liver specimens of congenital syphilitics at the Institute is claimed to be the first presentation of the kind to be recorded in literature. In a limited histopathological study of the liver biopsies of living infants with congenital syphilis, no pericellular fibrosis but degeneration of liver cells and evidence of chronic inflammation around the portal tracts were noted, unlike what was presented by Gault and Jayaraj in their autopsy case report.

(*xi*) Reference to congenital syphilis with renal or other visceral involvement did not appear in the studies undertaken. Comments on the dystrophy of hair, nails and other cutaneous appendages are omitted for brevity.

Observations were made in passing, on signs of tardive congenital syphilis occurring at an age period beyond the first two year period of life, *i.e.*, congenital syphilis when irremediable damage due to irreversible pathologic conditions are confronted with. Interstitial keratitis, Hutchinson's incisors, Moon's mulberry molars, bilateral nerve deafness, Clutton's joints, sabre tibiae, flattened bridge of nose with destruction of the nasal bones and septa, tabes juveniles, paresis juveniles and idiocy were touched on simply to emphasize the importance of preventive medicine through timely tackling of early congenital syphilis.

DIAGNOSIS AND MANAGEMENT OF SYPHILIS IN INFANCY

It was pointed out that in the experience of the speaker the diagnosis of congenital syphilis rested chiefly on (*i*) the relevant personal anamnesis and the obstetric history given by the female parent, (*ii*) to a certain extent on the results of interview through confrontation of the male parent and (*iii*) on the ensemble of the clinical, bacteriological, serological, roentgenological and occasionally the spinal fluid pattern and histopathological data obtained of the suspected infant or child.

The routine treatment prescribed is 150,000 units or ½ millilitre of PAM in oil (any brand that satisfies the W.H.O.

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Fig. 3

The Picture of a family high lighting the modes of syphilitic infection:

(*a*) The sexual highway leading to the infection of the central figure namely, the mother of the family who is infected but yet who does not present any manifest lesions on the surface of her body. The husband responsible for her infection is the 5th columnist and as such is out of the picture. Untreated syphilis in the mother was ultimately responsible for syphilis in her youngest offspring.

(*b*) The transplacental highway in syphilis *via* which the luetic infection of the once foetus, has now resulted in manifest syphilis in the live born innocent baby in arms.

(*c*) The non-sexual highway of syphilis has culminated in the occurrence of extra-genital chancres in the elder siblings standing on either side of the mother with (i) a chancre on the thigh, (ii) a chancre on the abdominal wall.

specifications) given daily, I.M., for 20 days for the light weight infants under three months of age or 3 lac units or 1 millilitre of PAM I.M., daily for 10 days for the older ones upto 2 years. This is not all. It is increasingly realised in recent years that the case of pre-natal syphilis of less than 2 years of age requires the attention of paediatric specialists. According to Moore, it is the paediatrician who takes care of the congenital syphilitic infants and children under the age of 14. The need for expert paediatric advice is indeed great in the management of the marasmic infant who requires fluids, salts and proteins for dehydration and blood for anaemia.

OTHER ASPECTS OF TREATMENT OF SYPHILIS IN INFANCY

In the series under review congenital syphilitic infants were hospitalised along with the mother. The treatment was completed within less than a month in practically every case except when the infected babies were discharged against medical advice or had died before completion of therapy. What a contrast to prepenicillin era—the days of metallic chemotherapy when the duration of therapy extended to nearly 2 years !

PROGRESS

(a) *Clinical.*—The clinical condition in early congenital syphilis showed marked improvement at the end of therapy. The cutaneous and mucosal lesions healed promptly ; no scars suggestive of rhagades were noticed in the series during the follow up period. The skeletal lesions subsided with relief from tenderness and pain. The visceral lesions showed signs of amelioration particularly in the liver series in which the size of the liver returned almost to normal limits ; jaundice disappeared.

(b) *Serologic.*—The positive serologic titre reverted to negativity or declined rapidly when the treatment was started within the first few months of the luetic infection in the infant. A delay in reversal of serology was recorded in infants for whom the treatment was started after 6 months of age.

(c) *Relapses.*—Clinical relapses in the experience of the speaker happened to be 1%.

(d) *Fatalities.*—It is confessed that in the presence of an overwhelming transplacental luetic infection, in a few infants as already pointed out deaths occurred in the early series despite desperate efforts. Fatal Herxheimer reaction has been noted by Holgen. In 1946-47, 10 deaths occurred among 59 cases administered commercial penicillin G bringing the mortality rate to 16·9%. In 1953, 3 deaths occurred among 102 cases treated with PAM and in 1955-57 one death among 26 cases treated with diamine penicillin.

Nevertheless the significant lesson to be learnt from the presentation of these disappointing notes in this retrospective study is that syphilis in infancy particularly early congenital syphilis is cent per cent preventable. It is the bounden duty therefore of every one concerned to take steps to see that syphilis in infancy is prevented. To this end we must at least recognise and adequately treat every syphilitic woman partner of the first generation in time with appropriate remedies. **The unborn innocents expect every one of us to do our duty!**

[In closing, a note of warning was issued on the possibility of mistaking acquired syphilis in infancy for congenital syphilis in infancy.

To clarify the issue of the subject of acquired syphilis in infancy—childhood, a photograph was projected presenting a woman with her congenital syphilitic baby on her lap and two older siblings, who, each one standing by the mother, had acquired an extra-genital chancre, the congenitally infected infant having donated the syphilitic infection through non-sexual contact to the two elderly girls.]

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Fig. 4

A marasmic infant in prenatal syphilis.

Fig. 5

The picture of an infant with cutaneous, mucous membrane and skeletal lesions in prenatal syphilis.

Fig. 6

Picture of an infant with ocular and nasal discharges teeming with treponema pallida.

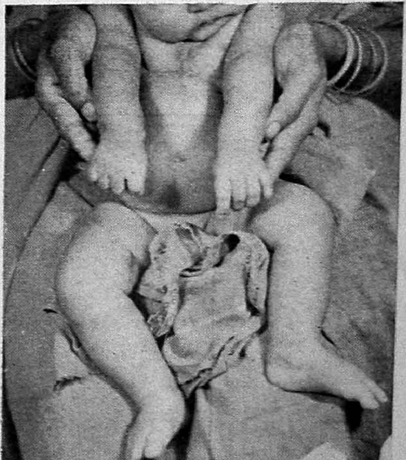


Fig. 7

Osteochondritis lower end of either forearm with pseudoparalysis.

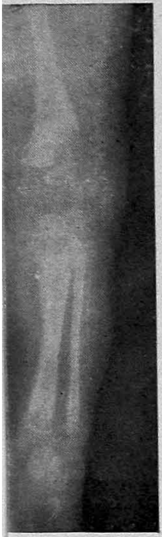
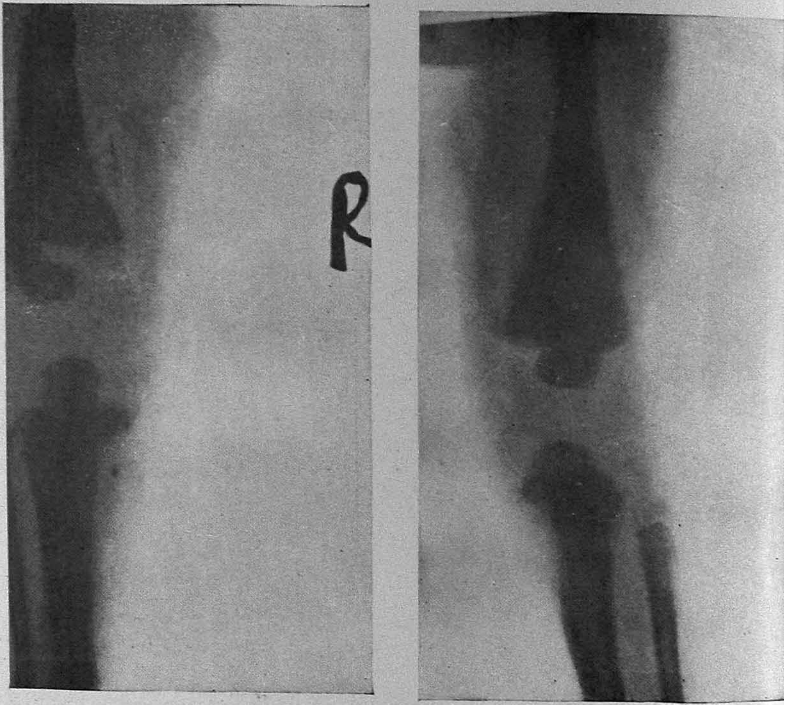


Fig. 8
m of the tubular bones
ıg the damage done by
ilis at various sites.



9
Skiagram of the bones of the lower limbs revealing the Wimberger sign, *i.e.* localised osteomyelitis producing a cat bite appearance in the upper and medial end of either tibia.

Fig. 10
Syphilitic pemphigus soles.

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Fig. 11
Exfoliation soles in a congenital syphilitic.

Fig. 12
Generalised cutaneous eruption in early prenatal syphilis with better localisation on the face.

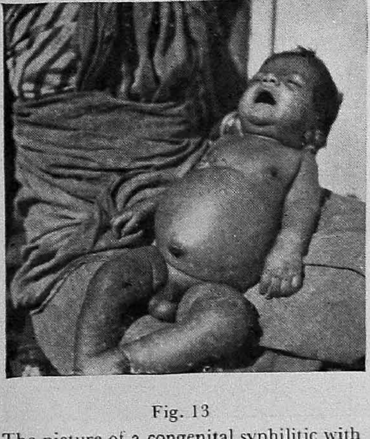

Fig. 13
The picture of a congenital syphilitic with a distended abdomen secondary to enlargement of the liver and the spleen.

Fig. 14
Bullous dermatosis—syphilitic pemphigus—the individual bullae have burnt—the bullous material revealed treponema pallida on dark-field illumination test.

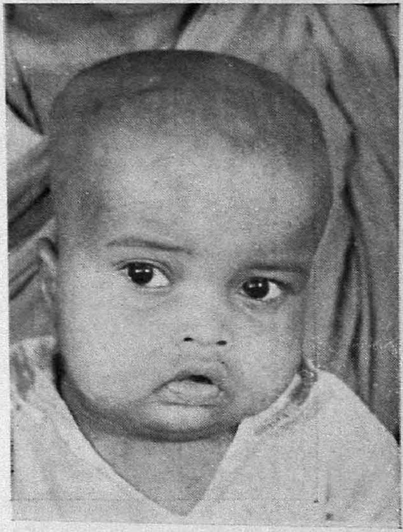

Fig. 15
Prenatal syphilitic with a prominent forehead.

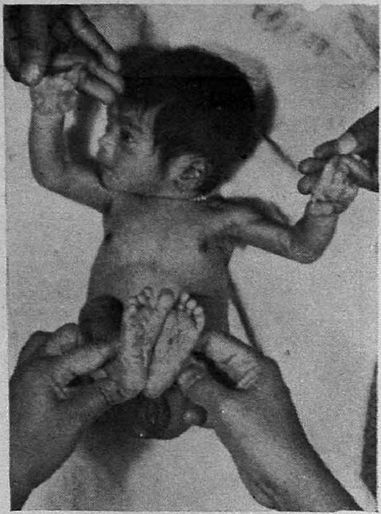

Fig. 16
Palmar and plantar lesions in a prenatal syphilitic.

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Fig. 17
Dactylitis in a prenatal syphilitic.

Fig. 18
Prenatal syphilitic with haemorrhagic lesions nose and mouth.

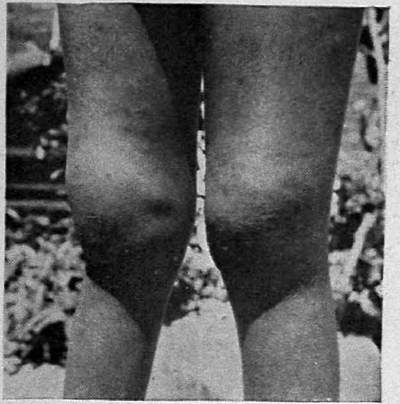

Fig. 19

Clutton's Joints—Painless symmetrical hydrarthrosis.

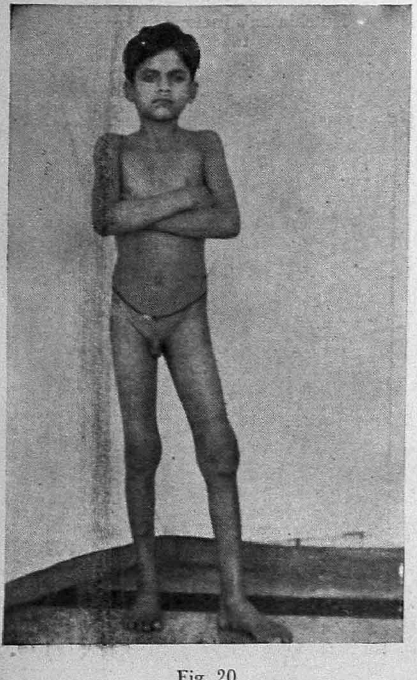

Fig. 20

Interstitial keratitis and Clutton's joints in a juvenile.

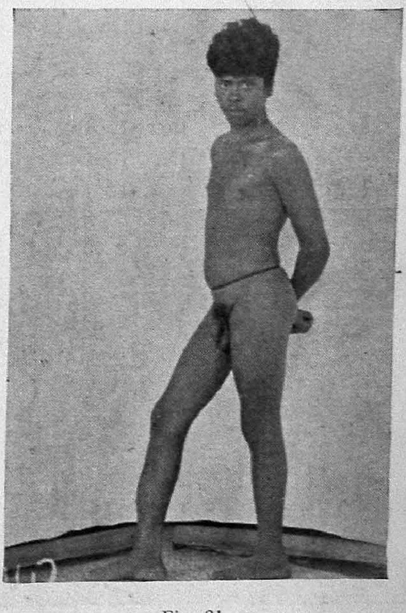

Fig. 21

Sabering of the tibia in a juvenile with prenatal syphilis (Late).

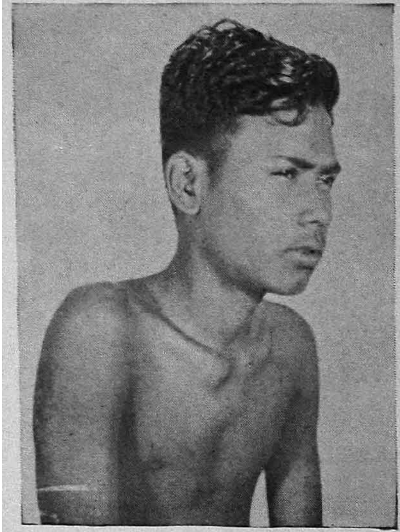

Fig. 22
The clavicular sign—the Higoumenakie's sign in prenatal syphilis.

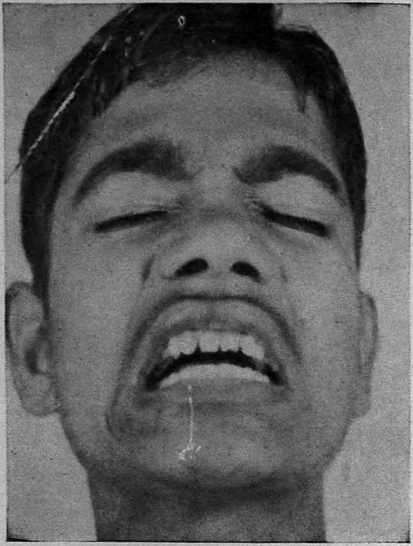

Fig. 23
Hutchinson's Incisors in juvenile tabetic.

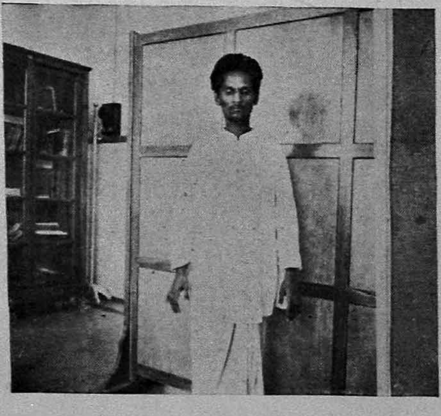

Fig. 24
A juvenile paretic.

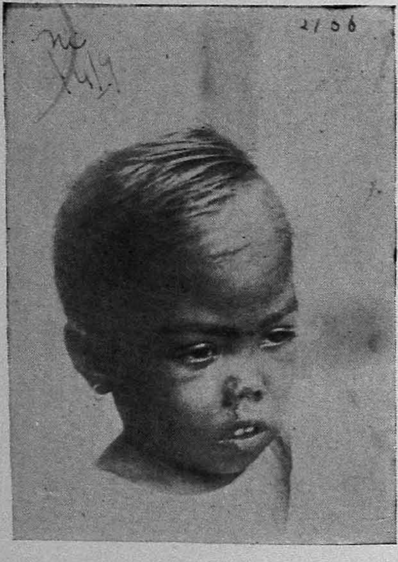

Fig. 25
Typical facies with flattened bridge of nose in prenatal syphilis.

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Syphilis and Infancy

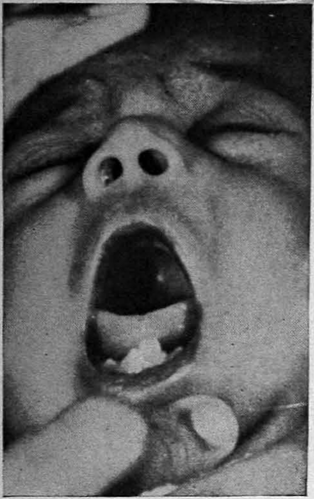

Fig. 26
An infant with prenatal syphilis born with teeth.

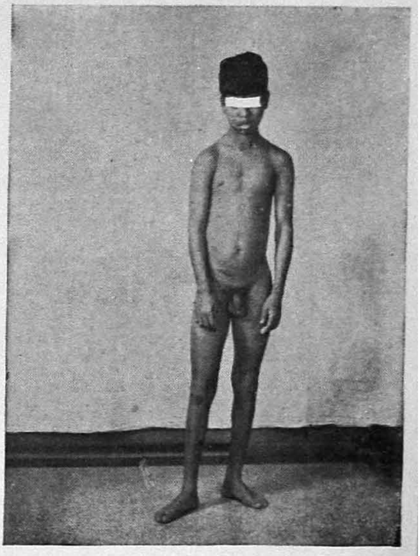

Fig. 27
Imbecility in a juvenile with prenatal syphilis.

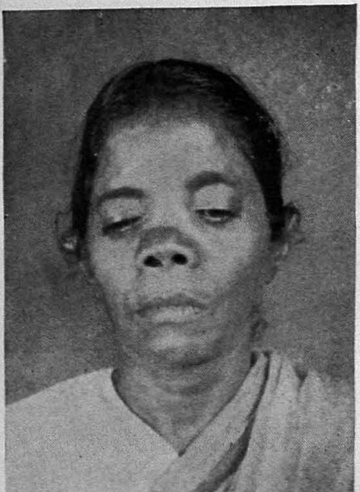

Fig. 28
The typical facies with flattened bridge of nose in prenatal syphilis.

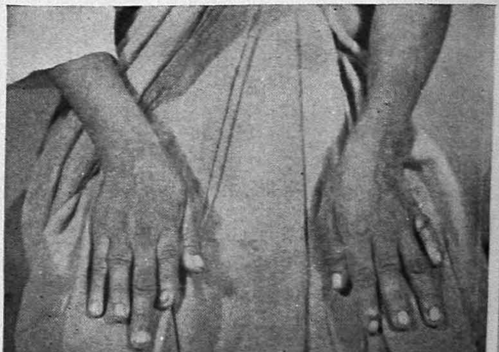

Fig. 29
Contracture of the little finger of the left hand in prenatal syphilis (Late).

Fig. 30

Acquired Syphilis in Infancy

Note the varying age periods of the lesions about the orifices and the ends of long bones.

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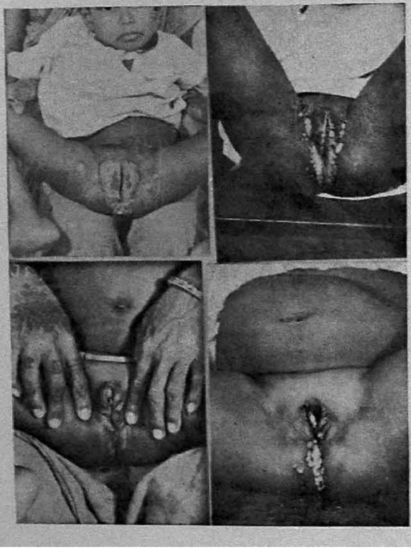
Fig. 31

Acquired Syphilis in infancy.



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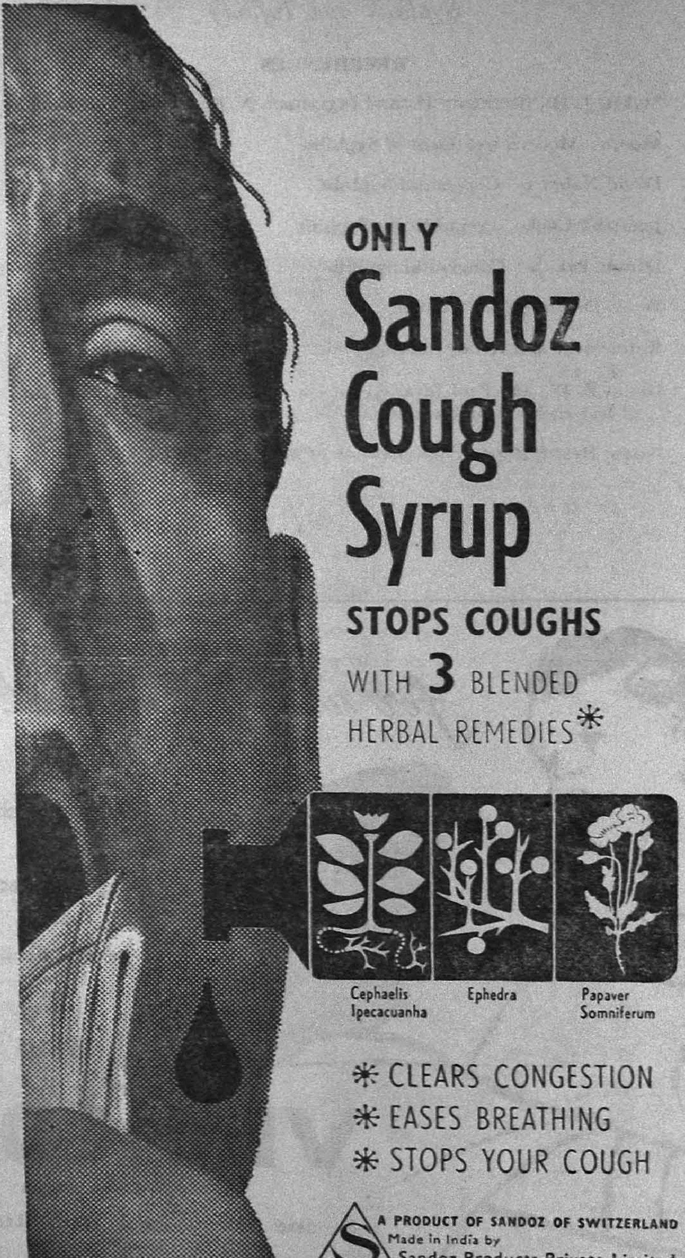
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Surgery in cardio vascular diseases

Dr. N. MADANAGOPALAN, M.D.*

Cardiac surgery has made rapid progress over the past two decades; and the factors concerned to attain the present status when we are able to correct certain cardiac anomalies, congential or acquired, to make the cardiac cripple a normal person at times and in other circumstances make the patient feel that his life is worth living have been:

(1) advances in anaesthesia

(2) introduction of hypothermia

(3) mechanical extra-corporial circulation.

Equally important has been the improvement in instruments and adjuvant devices for arriving at an accurate diagnosis.

Cardio vascular disorders which present with symptoms of cardiac insufficiency for which surgery is available, can be classified as follows :—

I. Surgery of vessels:

1. Patent ductus arteriosus.
2. Coarctation of aorta.
3. Aneurysms of thoracic aorta.
4. Vascular rings.
5. Transposition of great vessels.
6. Aortico pulmonary fistula.
7. Pulmonary arterio venous aneurysms.

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*Assistant Physician, Government Stanley Hospital, Madras.

8. Anomalous venous returns:

(*i*) Drainage of vena cavae into left side presenting as cyanotic heart disease.

(*ii*) Anomalous drainage of pulmonary veins on the right side into either vena cavae or auricle or ventricle causing pulmonary hypertension.

II. Surgery for valvular diseases:

1. Mitral stenosis.
2. Aortic stenosis.
3. Pulmonary stenosis.
4. Tricuspid stenosis.
5. Mitral insufficiency.
6. Aortic insufficiency.

III. Surgery for septal defects and complex developmental anomalies:

1. Tetralogy of Fallot.
2. Atrial septal defect.
3. Ventricular septal defect.

IV. Diseases of pericardium:

1. Constrictive pericarditis.
2. Possibly, tuberculous pericarditis even in a stage of effusion.
3. Malignancy.

V. For Coronary insufficiency:

For attempts at revascularisation of heart.

VI. Thrombo-embolectomy:

1. For pulmonary embolism.
2. Leriches syndrome—saddle thrombo-embolism at aortic bifurcation.
3. May be, for coronary thrombosis.

VII. For wounds and tumours of heart.

VIII. For cardiac arrest:

Use of defibrillator or electric pace maker.

PATENT DUCTUS ARTERIOSUS

The first successful surgical correction of patent ductus arteriosus was done by Gross in 1938.

The detection of this anomaly in childhood even without any symptoms is an indication for surgery because of hazards of this disease particularly bacterial endocarditis. Infection, physical or mental stunting or evidence of cardiac decompensation is considered as indication for operation at any age. Due to greater fragility of tissues and risks of rupture, operation is not indicated in older age group in patients with little or no cardiac enlargement and only minor cardiac symptoms referable to cardio vascular system. In presence of S. A. B. E. it is better to get over infection with antibiotics before operation is contemplated but poor response to antibiotic therapy is itself considered an indication for operation. A point to note is that operative procedures have shown that some cases diagnosed as P. D. A. on clinical grounds turn out to be aortico pulmonary septal defect or inter-ventricular septal defect with aortic regurgitation.

Methods.—1. Ligation.

2. Section.

3. Double ligation method.

Fixed rise in pulmonary vascular resistance is considered as contraindication to surgery as also presence of P. D. A. in association with other cardiac anomalies in which its presence may be nature's device to make the condition less crippling.

Coarctation of Aorta.—Strong pulse in upper extremity and weak or no pulse in lower extremity and delayed onset of femoral pulse are diagnostic signs of the disease. Retrograde aortogram is diagnostic.

The surgical treatment consists in excising the constriction and joining the two ends by direct suture or by means of aortic homograft. Other methods include 'By-pass operation' where the left subclavian artery may be turned down and united with the aorta at a point below coarctation.

Although it is considered by some that all patients with uncomplicated coarctation should have it repaired before third decade, presence of cardiac enlargement, significant hypertension, E. C. G. evidence of strain and cardiac failure are definite indications for surgical treatment and the younger the age when it is done, the better it is. Although persons in the older age group run a greater risk due to decreased vascular elasticity and associated atherosclerosis, old age group is not considered an absolute contraindication and successful results have been obtained in persons over age of 50 years.

ANEURYSM OF AORTA

Operations for saccular aneurysms of aorta are many and the important groups are :

1. Promotion of intrasaccular thrombosis.
2. External reinforcement.
3. Excision and aortorrhaphy or excision and aortic replacement by graft.

Insertion of wire into the sac in order to induce thrombosis is unsatisfactory, the risk is considerable and effective clotting cannot be guaranteed.

Wrapping the aneurysm in polythene cellophane causes an intense fibroblastic reaction which protects the sac from without, preventing further expansion and relieves pain.

Complete excision of saccular aneurysms with preservation or restoration of the aortic pathway is the ideal method and this can find a better place in descending thoracic aorta than the arch of aorta aneurysm. The continuity of the aorta after excision of the segment giving rise to aneurysm may be established by suture repair of the aortic wall, by plastic procedure upon the aorta or using a retained portion of the sac or by the use of a suitable homograft or a prosthetic tube.

VASCULAR RINGS

This may be the cause of dysphagia and dyspnoea due to compression of oesophagus and trachea. Bronchopneumonia is a common complication. The surgical treatment consists in breaking the constricting ring to release the pressure on the trachea and oesophagus.

TRANSPOSITION OF GREAT VESSELS

It may be considered the foremost cause of death due to congenital cardiac anomalies in infancy and also as the commonest cause of persistent cyanotic heart disease in the new born.

The surgical treatment consists of:

(1) Increasing the mixing of the blood flow between the right and left side by creation of an inter atrial septal defect and systemic-pulmonary anastomosis.

(2) Partial correction by anastomosis of Superior Vena Cava to the left atrium and right pulmonary veins to the right atrium.

(3) Right superior and inferior pulmonary veins to the right atrium and I.V.C. to the left atrium.

(4) S.V.C. and I.V.C. to left atrium and pulmonary veins to the right atrium.

Aortico-pulmonary septal defect.—This is often mistaken for P.D.A. and for repair of this defect hypothermia and some form of extra-corporeal circulation is necessary.

PULMONARY ARTERIO VENOUS ANEURYSMS

This may be the cause of central cyanosis due to admixture of venous blood from pulmonary artery with the oxygenated blood in pulmonary veins. The heart itself is normal but we may spot the continuous murmur over affected part of lung and a skiagram may show a rounded or irregular opacity which may be seen to pulsate on fluoroscopy and defined by angio-cardiography. Lesions may be single or multiple or bilateral. Treatment is by lobectomy or pneumonectomy and this is curative. Hence surgery is not possible when there are several widely distributed aneurysms.

ANOMALOUS VENOUS RETURNS

(a) *Anomalous drainage of both or either S.V.C. or I.V.C. into left atrium.*—Presents as central cyanosis with no obvious abnormal physical signs. In majority of groups with no symptoms no treatment may be necessary for the persistent channel or cure may be accomplished by simple ligation of that particular systemic vein that drains into left side, if other drains normally or at times, anastomosis of the anomalous vessel to the right atrium may have to be performed.

(b) *Anomalous pulmonary venous drainage.*—There may be total anomalous pulmonary venous drainage, all pulmonary veins draining into right side through either left innominate vein or superior vena cava or I.V.C. or persistent left S.V.C. or into right atrium itself.

In partial anomalous venous drainage one or more (more commonly right) but not all pulmonary veins drain into right atrium or S.V.C. or cononary sinus or left innominate vein or vena azygos or portal vein or other veins. It is of interest to note that sometimes the anomalous veins converge to form a chamber or sinus before they drain into systemic venous circulation.

Surgical treatment aims at anastomosis of the common pulmonary veins sinus to the left atrium in case of complete anomalous venous drainage. In partial anomaly, if patient is

asymptomatic with no cardiac enlargement or strain no surgery is indicated. If the patient is having symptoms referable to cardiovascular system, then attempt should be made to anastomose the anomalous channel to the left atrium.

A point to note is many cases of anomalous venous drainage have associated inter atrial septal defect.

Mitral Stenosis.—Significant block at the mitral valve produces pulmonary hypertension which can be alleviated by surgery. In general, any patient who is suffering from the effects of mitral stenosis requires commissurotomy but it is not an essentiality for those who are able to carry out their normal occupation without distress. The ideal case is the one with simple mitral stenosis and Grade II cardiac failure. Progression and intensification of symptoms are a strong indication for commissurotomy.

The definite contraindications are:

1. If predominant valvular lesion is mitral insufficiency or aortic insufficiency, (until such time these could be corrected by surgery).

2. It is not indicated during course of acute rheumatic activity and active S.A.B.E.

Although it is advisable to defer operation till age of 20—(ideal age group 20 - 50), it could be done in younger age if there are severe symptoms due to stenosis. Atrial fibrillation, previous embolisation and valvular calcification are no contraindications. Although commissurotomy may be deferred until after pregnancy, emergency commissurotomy may have to be done in pregnant women who present with severe pulmonary congestion due to tight stenosis and some are enabled to complete their pregnancy because of operation.

Surgical methods.—(1) Mitral commissurotomy: Objective is to enlarge the narrowed mitral orifice. The fused commissures

may be separated digitally or the dense adhesions may require use of special knife. The principle is that the split must be continued as far as the ring on both sides.

Other forms of surgical treatment include.—Relief of pulmonary congestion by (*a*) anastomosing the dorsal segment branch of the right inferior pulmonary vein to the azygos vein: (*b*) creation of an artificial atrial septal defect to provide an additional path of egress for the left atrial blood thus relieving pulmonary congestion (creation of Lutembacher's syndrome): (*c*) left auricular appendecectomy to prevent recurrent systemic embolism: (*d*) ligation of Inferior Vena Cava: with the double objective of relieving pulmonary congestion or heart failure and preventing pulmonary embolism secondary to phlebothrombosis in the legs in the cases with recurrent pulmonary embolism.

MITRAL INCOMPETENCE

There is no uniform procedure which has gained general acceptance and hence it can be said that operative treatment for this has not become well established. Attempts include,

(*a*) freeing the posterior leaflet from any adhesions to the shortened chordae or mural endocardium and elevating it to its proper position;

(*b*) use of constricting suture to decrease the size of atrioventricular annulus;

(*c*) creation of a trans-ventricular hammock by means of pedicled pericardial graft, and use of synthetic prosthesis have lost their supporters.

Paul Wood is emphatic that no operative treatment yet devised has been of the slightest benefit to cases of mitral incompetence.

ISOLATED PULMONARY STENOSIS

Operation is indicated if patient has exertional dyspnoea or fatigability or if right ventricular pressure exceeds 80–100 mm. of Hg. There is no indication for operation at present on acyanotic, asymptomatic patients with right ventricular pressure of less than 75 mm. of Hg. Ideal age is below twenty.

Surgical procedures available:

(1) Trans-ventricular valvulotomy—blind approach.

(2) Open valvuloplasty—under direct vision—using mechanical heart.

(3) Trans arterial approach with hypothermia.

AORTIC STENOSIS

There is the opinion that regardless of type of sürgical approach on the aortic valve whether by blind or direct vision technique, it is impossible to convert such a valve into a functioning valve without creation of aortic insufficiency.

Patients who are well compensated and are asymptomatic need not be subjected to surgery. This operation is contraindicated when there is marked insufficiency in association and if patients are beyond age of 50. The operation is indicated in both rheumatic and congenital aortic stenosis and associated coarctation of aorta is not a contraindication.

The patients for whom surgery is to be undertaken are those with severe stenosis with marked incapacity in form of exertional syncope, exertional angina or left ventricular failure.

Surgical procedures available:

(1) dilation of the valve by an instrument inserted through the left ventricle or through the aorta.

(2) permanent by-pass of aortic valve—a lucite tube placed in the apex of left ventricle, conducts blood through a modified Hufnagel valve and into the thoracic aorta.

AORTIC INSUFFICIENCY

The procedure which has found a recognised place is the insertion of Hufnagel valve into the aorta by multiple point fixation beyond the left subclavian artery. It is a rigid prosthetic plastic ball valve in descending aorta, preventing regurgitation of blood into left ventricle.

Recent interesting developments concern correction of aortic valvular incompetence by direct approach to the valve itself with excision of one cusp and narrowing of the aortic annulus to convert the valve into a competent bicuspid valve.

TETRALOGY OF FALLOT

Ideal age group for operation is from 3—12.

Surgical methods available:

1. Attempts at increasing the pulmonary blood flow and oxygenation of blood by anastomosing a systemic artery to a pulmonary artery.

   (*a*) Blalock—Taussig procedure—A subclavian or innominate artery anastomosed end to side (occasionally end to end) to the right or left pulmonary artery near its origin (creation of an artificial patent ductus).

   (*b*) Direct side to side anastomosis between the aorta and pulmonary artery (creation of aorticopulmonary fistula).

   These are only palliative operations.

2. It is now possible by means of open heart surgery to remove the stenotic process and to close the ventricular septal defect and thus to return the heart to a normal anatomic and physiologic state.

Present experience seems to suggest that the results of open operation are less satisfactory where a previous Blalock operation has been carried out and hence such preliminary treatment

is being avoided in those with minimal or moderate disability and where facilities for corrective operation are likely to be acquired later.

ATRIAL SEPTAL DEFECTS

The combination of pulmonary hypertension and a large left to right interatrial shunt are regarded as strong operative indications. But pulmonary hypertension with reversal of shunt is generally a contra indication.

Procedures available:

(*a*) Atrioseptopexy—Attempts at occluding the defect completely by suturing.

(*b*) Atrial well technique of Gross—In this a rubber well is created that could be sewed into the side of right atrium so as to permit the sewing together of rim of septal opening, working down through a pool of blood in the artificial well.

(*c*) Operation under direct vision.

Inter ventricular septal defect.—There is no indication to repair small ventricular septal defects in patients in whom the left to right shunt is small and there is no significant pulmonary hyper-tension. It has been found that with increased severity of pulmonary vascular resistance where the pulmonary flow is actually reduced and shunt is predominantly right to left, surgical closure is fatal.

Purpose of operation is to abolish the shunt by closure of defect. This can be repaired by either closed method or under direct vision.

Pericardiectomy.—The prognosis for patients with constrictive pericarditis is invariably poor and patient is a cardiac invalid if percardiectomy is not performed.

The purpose of pericardiectomy is the successful release of heart to increase its cardiac output. As a rule, the scar must be first removed from the left ventricle and only when we are sure that it is free to receive a greater amount of blood than previously the right ventricle must be decorticated. The pericardium is removed from as much of the anterior surface of ventricle as possible and it is not an absolute necessity to release the heart all around. The dangers of the operation are injury to the coronary arteries, phrenic nerve, tear of the myocardium and tearing of the thin walled right atrium.

There is a swing towards pericardiectomy being done even in stage of tuberculous pericarditis with effusion for fear of subsequent development of adhesive pericarditis.

Attempts at revascularisation of heart.—Several procedures have been used.

(*i*) Grafts of intercostal muscle, of omentum, of lung or of jejunum sutured to the heart to establish collateral circulation with these extracardiac vessels.

(*ii*) Cardiopericardiopexy: To create a sterile granulomatous pericarditis to promote pericardical vascular anastomoses with the coronary arteries.

(*iii*) Beck I operation :—

It consists of, (*a*) abrasion of epicardium and lining of parietal pericardium.

(*b*) application of an irritant to these surfaces.

(*c*) partial occlusion of the coronary sinus where it enters the right atrium.

(*d*) grafting of parietal pericardium and mediastinal fat to the surface of the heart.

(*iv*) Ligation of great cardiac vein with or without pericoronary neurectomy.

(*v*) Beck operation II.

Two stage operation :

(*a*) First - vein graft placed between aorta and coronary sinus for shunting arterial blood.

(*b*) Second stage - after interval of 2-3 weeks - coronary sinus partially occluded to raise the pressure in that vessel to cause the arterialised blood entering sinus from aorta flow retrograde back into the coronary capillaries.

(*vi*) Implantation of internal mammary artery in a tunnel in the myocardium.

(*vii*) Bilateral ligature of the internal mammary arteries below the cardiophrenic branches. It is considered that the value of this procedure mainly lies in its simplicity and not in its efficacy. Recent studies seem to reveal that the same result could be obtained following simple incision of the chest wall without doing anything to the internal mammary arteries and without any attempts at alteration of the coronary circulation.

Coronary endarterectomy.—The frequent occurrence of atherosclerosis limited to first few cms. of the coronary arteries has given scope for attempts to remove the occlusive material by endarterectomy. Where fibrotic changes are encountered in the small radicals of coronary arteries poudrage is performed as an alternative to coronary endarterectomy.

SUMMARY

An attempt has been made in this article to discuss the place of surgery in heart diseases, congenital or acquired.

ACKNOWLEDGMENT

I must thank Dr. S. Panchanadam, M.B., B.S., for helping me in preparing this article.

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Management of Lepra Reactions

Dr. S. Z. AHMED, M.B.B.S.*

The condition known as Lepra Reaction or 'Erythema Nodosum Leprosum' is very frequently seen during treatment of leprosy with sulfone drugs. It is due to the breaking down of bacillary clumps and liberation of bacillary proteins and bacillary toxins which cause an allergic response. The body is sensitised, as it were, to the presence of the foreign invader. This condition is reminiscent of Jarish Herxheimer reaction in syphilis during arsenical treatment. There are several ways of treating this condition, but the first and foremost thing is to stop sulfone treatment and then put the patient on one of the following remedies :—

1. *Pot. Antimony Tartrate.*—This is given as an intravenous injection every alternate day. The dose is usually 0·04 gramme in 5 c.c. of distilled water. The solution has to be prepared fresh just before giving the injection as it is likely to deteriorate on standing. About six injections are generally sufficient to bring down the temperature. If P.A.T. is not available, Fouadin (Bayer) 2 c.c. intramuscularly is given every other day for a maximum of six injections with equal benefit.

2. *Prednisolone.*—This is generally given as tablets of 5 mg. orally. On the first day and subsequent two days, one tablet is administered sixth hourly followed by 3 tablets daily on the next 3 days. Then 2 tablets daily for another 3 days, thus gradually reducing the dose to 1 tablet daily which should be continued for a week and then reduced to half a tablet daily for another week. Patients are found to tolerate sulfones better under cover of prednisolone. So when one tablet is being taken,

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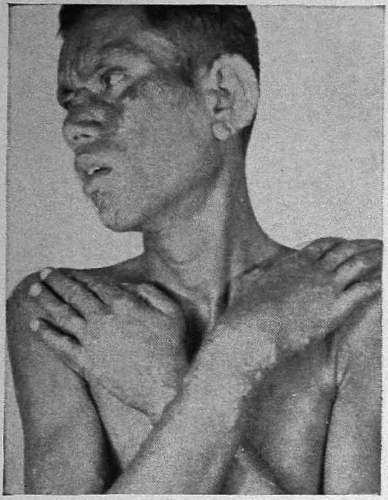
we re-commence sulfone therapy, with a small dose say 25 mg. a day and we gradually withdraw prednisolone, simultaneously increasing the Sulfone.

3. *Periston 'N'* (*Bayer*) or Polyvinyl Pyrrolidone available as 100 c.c. vials, is given as a slow intravenous drip. The solution need not be diluted further by adding saline or glucose saline. It is to be given once daily for 8 days and the reaction subsides remarkably. It can be given concurrently with Prednisolone orally in resistant cases.

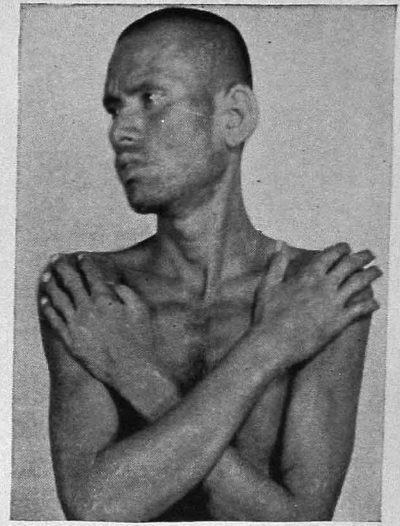
4. *Mercurochrome.*—1% Solution (Aqueos) is administered intravenously in subacute and chronic cases of Lepra Reaction where the patient has fugitive swellings of the hands and feet with either low temperature or normal temperature. The dosage is 5 c.c. to begin with, given intravenously every other day, gradually increasing by 1 c.c. to a maximum of 7 to 8 c.c.

Vitamin B-12 is used parenterally during convalescence from reaction for its general tonic effect and also to counteract the anaemia so often produced by sulfone therapy. This is given in doses ranging from 100 microgrammes to 500 microgrammes daily.

Acute Leprous Neuritis or 'Nerve Reaction' is usually an accompaniment of Acute Lepra Fever or may occur by itself as a single presenting symptom. It causes excruciating pain along the superficial nerves, *viz.*, ulnar, peroneal and great Auricular, due to pressure of granulomatous deposits under the nerve sheath. This condition may be treated by (*a*) injections of calcium gluconate 10 c.c. intravenous daily and or (*b*) Prednisolone orally in similar dose as for general reaction. Locally it is advisable to give injections of 0·5% sodium carbonate with 10 minims of adrenaline 1 in 1000 around the nerve, or inject Hyalmanidose into the nerve. Deep X-Ray therapy is given locally to the affected nerve to reduce inflammatory swelling. At a later stage, a decapsulation of the nerve may be necessary to give permanent relief from pain.

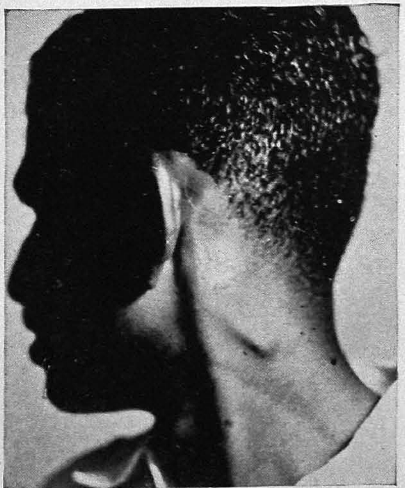


Before treatment.



After treatment.

Acute Lepra Reaction treated with Prednisolone.



Acute Leprous Neuritis (Nerve Reaction).

Acute Leprous Iritis or 'Eye Reaction' is another condition seen during the stage of Acute Lepra Reaction or may occur by itself as a sole manifestation. This is caused as a result of lodgement of bacillary emboli in the iris muscle. It produces secondary conjunctivitis with photophobia and lacrymation. Treatment is to instil prednisolone eyedrops in the day time and apply prednisolone ointment at night time for continuous effect. In severe cases, subconjunctival injections of 1% Hydrocortisone in normal saline may be necessary to reduce the inflammation.

Acute Rhinitis is also an acute complication of sulfone therapy. The patient is unable to breathe and suffers greatly due to blocking of the nasal passages by swelling and congestion of the nasal mucosa. Treatment is to instil Prednisolone nasal drops or spray the solution in both the nares 4 to 6 times daily and it is found to give relief.

In conclusion, corticosteroids have taken the place of antimonials in the treatment of Lepra Reaction and its several manifestations, just as sulfones have replaced hydnocarpous products in the specific treatment.

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Haemorrhagic Disorders*

Dr. A. GAJARAJ†

Bleeding diatheses form a group of diseases of widely differing etiology, but have in common an abnormal tendency to bleed. Excessive bleeding is due to a breakdown of the haemostatic mechanism.

Hence an introduction regarding the mechanism of haemostasis will be of great assistance in understanding the derangements of the process.

Mechanism of Haemostasis.—Constitutes two main stages that of initiation and maintenance.

Initiation.—Immediately following injury, the injured blood vessel undergoes a temporary reflex vaso-constriction. The platelets next agglutinate to the intima and form a plug at the opening of the damaged vessel. The platelets also liberate a constrictor substance, serotonin which causes constriction of the blood vessels at the site of injury and maintains the initial vaso-constriction of the nervous reflex.

Maintenance is achieved by the formation of clot, its contraction and organization.

Extravascular factors influence the mechanism. Tissue tension and compression of blood vessels by the escaped blood lessens blood loss. In poorly supported loose tissue (*e.g.*, the orbit, nose) bleeding is more.

The mechanism of coagulation requires consideration. It is a complex phenomenon involving a chain of reactions between at least 9 factors (all present in the blood).

*Presented at the Weekly Clinical Conference, Government General Hospital.

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The basic fact of the mechanism was devised as long ago as 1904 by Morawitz. Though it remains the same, research work since 1940 has proved it to be anything but simple. To express in the simplest form, clotting is brought about in 3 stages:

(1) formation of substances called thromboplastin from the interaction of platelets and certain factors in the plasma.

(2) action of thromboplastin converting the soluble inactive prothrombin in the presence of certain factors, to thrombin.

(3) action of thrombin on a soluble fibrinogen to form fibrin.

This simple 3 stage mechanism sounds complex because each one of these stages is governed by the presence or absence of certain factors. Taking them one by one we shall deal first with thromboplastins. Two types are described, (i) plasma thromboplastin (ii) tissue thromboplastin.

Plasma thromboplastin is formed in the blood by the interaction of platelet factors, calcium ions and probably five or more plasma factors.

They are:—

(1) Anti haemophilic globulin factor—Synonym factor VIII-A. H. G.

(2) Christmas factor (CF) synonym—Plasma thromboplastin component or PTC; factor IX (Quick).

(3) Stuart factor SF.

(4) Hageman factor (HF).

(6) Ac globulin—Proaccelerin; labile factor: factor V.

(7) Power factor PF.

According to Quick, AHG and CF are the two which have been most clearly established.

Mechanism of haemostasis - Initiation - Vascular.
Maintenance - Coagulation.

Mechanism of coagulation.—Stages and factors-tabulation.

(1) Formation of thromboplastin → Plasma

- (1) AHG Anti Haemophilic Globulin factor - Synonym factor VIII.
- (2) Christmas factor (CF) Synonyms - plasma Thromboplastin component or PTC; factor IX (Quick).
- (3) Stuart factor SF.
- (4) Hageman factor (HF).
- (5) Ac globulin :— Pro-accelarin; labile factor; Factor V.
- (6) Plasma Thromboplastin antecedent; PTA.
- (7) Power factor PF.
- (8) Ca.
- (9) Platelets.

Stage I

– Tissue

- (1) Factor V Ac Globulin Proaccelerin; labile factor.
- (2) Factor VII tissue Thromboplastin; Pro-convertin stable factor.
- (3) Stuart factor (SF).
- (4) Power factor (PF).
- (5) Calcium.

(2) Convertion of prothrombin to thrombin

- (1) Labile factor Ac Globulin or proaccelerin; factor V.
- (2) Stable factor proconvertin; factor VII.
- (3) Stuart factor.
- (4) Power factor.
- (5) Calcium Ions.

Stage II

(3) Convertion of Fibrinogen to Fibrin.

(4) Inhibition of coagulation—

(1) Anticephalin.
(2) Heparin.
(3) Thrombin adsorption by fibrin.
(4) Fibrolysin.

Tissue thromboplastin is not the same as plasma thromboplastin. It is formed from blood vessels and tissues as a result of injury. It has also been extracted from tissues like brain and lung. This tissue thromboplastin interacts with certain factors and forms prothrombinase which has action similar to plasma thromboplastin.

The factors required for this convertion of tissue thromboplastin to prothrombinase are :

(1) Factor V (Ac Globulin, Proaccelerin ; labile factor).
(2) Factor VII tissue thromboplastin ; proconvertin or stable factor.
(3) Stuart factor (SF).
(4) Power factor ; PF and
(5) Calcium.

Conversion of prothrombin to thrombin - 2nd stage is by the action of plasma thromboplastin or prothrombinase of tissue Thromboplastin, perhaps both and several factors of plasma.

They are :—

(1) Labile factor (Ac Globulin or proaccelerin ; factor V).
(2) Stable factor (procenvertin ; factor VII).
(3) Stuart factor.
(4) Power factor.
(5) Calcium Ions.

ACTION OF THROMBIN THIRD STAGE

(1) It converts fibrinogen to fibrin.

(2) It catalyses in some manner the production of the plasma thromboplastin.

Inhibition of coagulation.—In addition to factors for the mechanism of clotting, there are also factors in the blood which inhibit or antagonise its formation. Coagulation can therefore be said to occur when the rate of production of clotting factors exceeds the rate at which they are neutralised by inhibiting agents. Inhibiting factors are (1) anticephalin: neutralises limited amounts of thromboplastin (2) heparin: acting along with heparin cofactor (which is perhaps the same as anti-thrombin) :—(a) interferes with the production of thromboplastin (b) inhibits reaction of thrombin on fibrinogen (c) converts thrombin to an *inactive* meta-thrombin (3) thrombin itself is removed by adsorption by fibrin. (4) fibrolysin or plasmin is formed in the blood in certain conditions from inactive plasminogen which lyses fibrin.

CLASSIFICATION OF HAEMORRHAGIC DISORDERS

Having gone through the chain of incidents that take place in the mechanism of haemostasis, it is important to realise that a break or defect at any level in the chain, congenital or acquired is apt to produce bleeding diathesis. The defects may be :

(1) vascular.

(2) platelet abnormalities.

(3) coagulation abnormalities.

(4) hypo-prothrombinaemic states.

(5) fibrinogen deficiency.

(6) fibrinolysis.

(7) coagulation inhibitors.

(1) Vascular—which again may be acquired or congenital.

Congenital—Telangiectasia. Hereditary Haemorrhagic Telengiectasia (Osler-Rendu-Weber Disease). Vascular pseudohaemophilia (Von Villebrand Disease). Ehlers Danlos Disease.

Acquired—*symptomatic vascular* (non - thrombocytopenic) purpuras due to infection, drugs, nutritional (scurvy) and metabolic disorders like uraemia, diabetes and Cushing's disease; systemic vascular diseases like hypertension, arteriosclerosis, periarteritis nodosa, and, amyloidosis - allergy.

Dysproteinemias—cryoglobulinaemia, benign purpura haemorrhagica and macroglobulinaemia, multiple myeloma.

Henoch Schonlein purpura (anaphylactoid), miscellaneous vascular disease like senile, simple, easily bleeding, purpura simplex; mechanical, orthostatic purpura and fat embolism.

(2) *Haemorrhagic disorders to platelet abnormalities.*—These may be—

(1) Quantitative—thrombocytopaenia.

(2) Qualitative—thrombocytoasthenia.

Thrombocytopaenia may be (i) primary (ii) secondary.

Primary: may be congenital or idiopathic, essential thrombocytopenia, purpura haemorrhagica.

Secondary to drugs, chemicals, leukaemia, aplastic anaemia, bone marrow infiltrations by malignancy, myeloma etc. multiple myeloma; myelo sclerosis, malignant lymphomas. disseminated lupus erythematosus; hyper-splenism. Infection, megaloblastic anaemia, thrombotic thrombocytopenic purpuras. In-compatible and massive blood transfusions; food allergy, haemangiomas; haemolytic diseases of the new born.

(3) *Haemorrhagic Disorders due to coagulation abnormalities.*—Deficiency of the plasma clotting factors: (1) Antihaemophilic globulin. AHG. *i.e.* factor VIII - Haemophilia 'A'. Classical Haemophilia; alpha Haemophilia; 7 times more common than

the Haemophilia 'B'. (2) Christmas factor C. F. Factor IX - Haemophilia 'B' plasma thromboplastin component deficiency, Beta Haemophilia. (3) Plasma thromboplastin anticedent - PTA deficiency. Haemophilia - C. (4) Hageman factor deficiency (Hageman trait).

(4) *Hypo-prothrombinaemic states*: due to deficiencies of (1) prothrombin (2) Stuart factor (3) *Factor V* (4) power factor (PF) (5) *Factor VII.*

Congenital deficiencies are rare (para haemophilia) and usually one factor is deficient; acquired deficiencies are much more common and usually involve several factors. (1) Deficiencies of Vitamin 'K' (2) Liver diseases (3) Over-dosage with anti-coagulant drugs or the commarin and indandione groups; Dindivan Group; (4) miscellaneous disorders like tuberculosis and disseminated malignancy; severe infections.

(5) *Fibrinogen deficiency - synonyms.*—Hypofibrinogenia, fibrinogenopenia. Normal concentration is 200 to 400 mg/100 cc. plasma. Bleeding occurs when it is less than 60 mg. to 80 mg%. May be acquired or congenital, usually acquired. Severe liver diseases, metastatic carcinoma, tuberculosis, polycythaemia and defibrination syndrome, where there is intravascular clotting due to thromboplastin. This is seen in incompatible blood transfusions, certain obstetric disorders and major surgical procedures.

(6) *Fibrinolysis.*—Excessive fibrinolysis due to abnormal activation of the fibrinolytic system. Inactive plasminogen is converted to plasmin. Seen in defibrination syndrome, extensive trauma, burns, acute leukaemia, liver damage, disseminated carcinoma especially prostate.

(7) Natural coagulation inhibitors seen in (1) pregnancy (2) lupus erythematosus, malignancy, malignant lymphomas. (3) Apparently no cause, (4) as a result of replacement therapy in haemophiliacs. They act at the level of thromboplastin formation usually, less commonly at other stages also.

PRACTICAL INVESTIGATIONS THAT ARE DONE IN THE HAEMORRHAGIC DISORDERS

The stage in the chain of mechanism of haemostasis, at which a certain defect is responsible for a bleeding disorder can be determined by some practical investigations.

Some of these are routine *viz.*, (1) capillary resistance test of Hess (2) platelet count (3) clot retraction test (4) bleeding time (5) whole blood coagulation time.

In addition there are some special tests which need elaboration. They are (1) prothrombin time (one stage prothrombin test) (2) prothrombin test (Two stage prothrombin test) (3) prothrombin consumption test (4) thromboplastin generation test (5) fibrinogen estimation test.

Thromboplastin generation test.—This measures the amount of plasma thromboplastin formed.

One stage prothrombin test: determines the time taken by plasma to clot after tissue Thromboplastin (usually brain extract has been added to it under standard conditions). It detects moderate to severe deficiency of a number of clotting factors such as factors V, III, SF, PF, Prothrombin and Fibrinogen, and also some coagulation inhibitors by a modification of the test.

Two stage prothrombin test.—Here prothrombin is first converted to thrombin and then measured as such. It gives a good measure of the prothrombin.

Prothrombin consumption test.—When normal human blood clots almost all the contained prothrombin has disappeared from the serum in one hour. The amount of prothrombin consumed depends on the amount of plasma thromboplastin formed. Therefore the test is impaired in disorders *e.g.* (1) the 1st stage of coagulation (formation of thromboplastin); (2) deficiency of soluble clotting factors; (3) deficiency in the number of or function of the platelets; (4) presence of coagulation inhibitors. This is a useful test but not as sensitive as thromboplastin generation test.

Fibrinogen estimation.—Thrombin is added to blood suspected to be deficient in fibrinogen and to a sample normal blood also. In fibrinogenopenia blood takes much longer time to clot.

LABORATORY DIAGNOSIS

The first step is to determine whether a patient has a coagulation defect or a capillary abnormality with or without thrombocytopaenia.

Preliminary investigations constitute a platelet count, bleeding time, tourniquet test and whole blood coagulation time.

Whole blood clotting time → normal - vascular abnormality
- platelet abnormality - qualitative quantitative

↓

Abnormal

One stage prothrombin test abnormal	*One stage prothrombin test normal*
(1) Fibrinogen deficiency	(1) Classical haemophilia (AHG) plasma factor VIII deficiency.
(2) Prothrombin deficiency	(2) Christmas disease factor IX serum deficiency.
(3) Factor V deficiency (Plasma)	(3) P.T.A. deficiency.
(4) Factor VII deficiency (Serum)	(4) Circulating anticoagulants.

(1) If only coagulation time were normal the defect were in the capillaries or platelets and can be differentiated by the platelet count, bleeding time, capillary resistance test, bone marrow studies and other specific tests depending on the primary conditions, in secondary purpuras.

(2) If the coagulation time is prolonged, the patient definitely has a coagulation defect. This test is unfortunately not sensitive and many patients with clotting defects give normal time.

Clotting defects may for convenience be divided into two groups on the basis of one stage prothrombin test.

(1) In which one stage prothrombin test is defective. This group includes deficiencies of one or any combination of fibrinogen, prothrombin, factor V and factor VII.

(2) 2nd group—One stage Prothrombin test is normal and includes Haemophilia, christmas disease, cases of circulating anticoagulants.

GROUP WITH ABNORMAL ONE STAGE PROTHROMBIN TEST

Includes deficiencies of:—

(1) Fibrinogen; (2) prothrombin; (3) Factor V; (4) Factor VII. They are differentiated by the following features:—

Fibrinopenia.–Plasma may not clot at all, or a very small, friable wispy clot may form. If thrombin is added to patient's blood and normal blood, patient's blood does not clot.

Prothrombin.—Deficiency can be detected by two stage prothrombin test where prothrombin is converted into thrombin and assessed.

Factor V.—To detect this add aluminium hydroxide both to normal plasma and to patients plasma, and contrifuge. This adsorps prothrombin factor VII and factor IX or Christmas factor and will be at the bottom. The supernatent plasma in the case of normal plasma will contain factor V and anti-haemophilic globulin, whereas patient's plasma, may not contain factor V. Now if 10% of supernatent normal plasma is added to patient's plasma, the one stage Prothrombin test gets shortened.

Factor VII.—Normal serum contains factors VII and CF but not factor V Proth or AHG. Then if factor VII is deficient, on adding 10% of normal serum to patient's, one stage prothrombin test will be shortened.

Group with normal one stage prothrombin time.—Diagnosis rests between (1) haemophilia (2) Christmas Disease and (3) presence of circulating anti - coagulants. Thromboplastin generation time is abnormal in all.

Haemophilia.—The AHG is lacking. This is normally present in the plasma. Hence when it is lacking plasma of patient treated with aluminium hydroxide will not form thromboplastin.

Christmas Disease due to lack of factor IX which is normally extracted in the serum. Thus the patient's serum is ineffective in thromboplastin formation.

Circulating anticoagulants.—Inhibit thromboplastin formation by inhibiting the action of both antihaemophilic globulin and christmas factors. Thromboplastin generation test is abnormal in general.

DIAGNOSIS AND MANAGEMENT OF A HAEMORRHAGIC STAGE

The initial diagnosis of a haemorrhagic state is usually made because of the excessive bleeding from small injuries, epistaxis, oozing from gums, menorrhagia, gastro intestinal haemorrhage, haematuria, easy bruising of purpura, undue bleeding after a tooth extraction or surgical operation. On the contrary, a patient may complain of symptoms which he does not connect with abnormal bleeding such as deep tissue swellings and painful or swollen joints.

Pointers which confirm the existence of haemorrhagic state are affections of different parts of the body, a long history of abnormal bleeding especially if existing from childhood and a history of similar instance in one or more blood relations. Points against will be limitation of bleeding to some particular

site and the presence of features suggesting an organic cause for haemorrhage.

Before calling a condition as a bleeding disorder care must be exercised. Even normal people may ooze for a long period after, say, a tooth extraction even for 24 hours. But it is probably abnormal for haemorrhage to continue beyond 48 hours unless a local cause such as acute sepsis is responsible. Any bleeding continuing for more than 24 hours should be regarded with suspicion. Any patient requiring blood transfusion after a tooth extraction is probably suffering from bleeding diathesis which includes leukaemia.

In taking the history it should cover all major haemorrhagic episodes and results of injuries or surgical operations.

Hereditary Haemorrhagic Diathesis is one common cause of bleeding. It is not enough just to ask for bleeding abnormality in the family. A family tree should be drawn going back to at least the patient's grand-parents.

If the patient is a male and if only male members of the blood relations are affected while females are normal, the condition probably is haemophilia or christmas disease. If both males and females are affected it is perhaps an inherited defect of another type. An extremely rare exception is the occurrence (of homozygous females) in haemophilic families as the result of marriage of a male haemophilic with female carrier.

Factor VII and factor V deficiency appear to be inherited as non sex-linked, and probably dominant factors. Fibrinopenia is inherited as a recessive character in cousin inter-marriages. PTA deficiency may be inherited as a Mendelian dominant.

If haemorrhagic state has existed from infancy it is likely to be hereditary, even if there is no familial history. In 25% it can arise '*de novo*'. However in the absence of a family history it is difficult to differentiate haemophilia and christmas disease on the one hand and deficiency of factors V and VII, or fibrinogen on the other.

Acquired.—If there is no family history and the condition has arisen in late childhood or adolescence or adult life it is unlikely to be hereditary and an attempt should be made to discover some immediate cause.

Circulating anticoagulants inhibiting the formation of thromboplastin may give a haemophilia like state, *e.g.*, during or after pregnancy, foetal, maternal antigenic incompatibility etc.

An idiopathic deficiency of anti-haemophilic globulin factor has occurred as an acquired condition in both sexes and consequent bleeding abnormality.

Factor V deficiency may occur as a complication of severe infections, liver diseases etc.

Fibrinogen deficiency may occur if there is severe liver disease, actual fibrinolysin or entry of tissue fragments into the circulation (mentioned already).

CLINICAL DIFFERENTIATION

Some clinical differentiation can be made between the haemorrhagic disorders.

In general, spontaneous bleeding, such as oozing from the mucous membranes and the occurrence of petechiae or ecchymoses in the skin suggests the group of disorders due to vascular defects. Only rarely is there bleeding into muscles and internal organs. In defective coagulation the bleeding is not limited to the skin and mucous membrane and is usually related trauma or tissue damage. When it affects the body surface, obvious haemorrhage occurs usually in the form of a slow and persistent oozing which may continue for many days and weeks. If there is no break on the surface, persistent bleeding into the tissues may produce haematomas sometimes of an enormous extent and producing pressure symptoms. Some of the manifestations are haemarthrosis, haematuria unrelated to trauma or infections.

Thrombocytopenia may be primary or secondary. The primary may be congenital or idiopathic. Secondary purpura

is more common and results from a number of conditions listed earlier. It is characterised by lymphadenopathy, splenomegaly, bone tenderness fever and anaemia, out of proportion to blood loss. The ESR is raised. In idiopathic thrombocytopenic purpura, anaemia (degree) depends on the blood loss.

Haemorrhagic Telangiectasia (Osler Redu-Weber Disease) may be observed over the lips, tongue, fauces or in nose and sometimes in the skin particularly around finger nails. These can be differentiated from petechial haemorrhages by the blanching produced on pressure.

Vascular pseudo-haemophilia.—(Von Willebrand's Disease) affects both sexes and inherited as a Mendelian dominant. The bleeding time is prolonged.

Ehlers Danlos Disease is characterised by a deficiency of the formation of collagen which prevents firm healing and causes undue friability of the blood vessels. There is hyperextensibility of the joints and stretching scar tissue.

Henoch Schonlein or anaphylactoid.—(hypersensitivity reaction) presents a purpuric rash. *Joing* abdominal and renal manifestations. The rash is characteristically distributed over the buttocks, on the backs of the elbows and extensor surfaces of the arms, lower leg, ankle and foot and usually bilateral. They occur in recurrent crops which progressively fade in two weeks.

Treatment of haemorrahagic disorders.—Due to coagulation defect.

This is far from satisfactory. No general treatment other than transfusion of normal blood or of certain blood derivatives has given any encouragement. Rational treatment must aim at correcting the basic coagulation defect. The missing factor must be replaced and its normal production in the body stimulated. In the case of circulating anticoagulants these must be removed or their production inhibited.

At present replacement therapy is by transfusing normal blood or by injecting certain purified fractions of normal blood having the required factor.

Stimulation of the production of such factors by the body has not yet been achieved. Only one type of anti-coagulant heparin can be removed from the blood in *vivo*. Cortisone and ACTH may diminish the production of anticoagulant, anti-bodies but available evidence is scanty.

Various preparation of tissue thromboplastin etc., have not proved satisfactory.

General treatment by replacement is used only for acute episodes or anticipated minor surgical procedures. In the treatment of haemophilia and factor V deficiencies, the blood or plasma should be fresh preferably less than 24 hours old as the anti-haemophilic globulin factor and factor V are unstable. Care must be taken to avoid any clot when collecting blood. Clot also destroys AHG.

Christmas factor is a stable factor and so christmas disease replacement can be done by stored blood or plasma. The same holds good for deficiencies of factor VII, PTA, fibrinogenopenia.

Local treatment—consists of pressure applied for a few minutes in conjunction with a suitable coagulant, since it allows time to form a firm clot in the mouths of the damaged vessels. Tight pressure should be avoided.

Local applications that have been found useful are fibrin foam, oxidized cellulose or gelatin sponge. These can be left in site until they are digested. Russel Viper's venom and thrombin can also be used.

In fibrinopenia the local application of thrombin or russel Viper venom are useless unless continued with a concentrated solution of fibrinogen. Even then the clot formed is not satisfactory as it does not extend into the damaged vessel.

(2) *Haemorrhagic telengiectasia.*—(Osler Redu - Weber Disease). In addition to the symptomatic management large doses of oestrogens are useful 0·25 mg. of ethinyl oestradiol daily in males methyl testosterone (5 mg. daily) is also given to prevent undesirable feminizing effects.

(3) *Vascular pseudo haemophilia.*—Apart from symptomatic treatment adreno cortical steroids may be tried to relieve an episode of troublesome bleeding, but in general results are disappointing. The tendency to bleed decreases with age.

(4) *Symptomatic vascular purpuras.*—Here treatment of the underlying cause is essential and withdrawal of offending agents like drugs.

(5) *Henoch Schonlein Purpura* is a self limiting disease. Treatment is symptomatic. Anti-histaminics lessen exudation. Adreno-cortico steroids are of help in patients with severe symptoms.

(6) *Idiopathic thrombocytopenic purpuras.*—the main therapy is steroid administration and splenectomy. In addition fresh whole blood transfusion is an important measure in patients with severe bleeding. Platelet transfusions are indicated in certain cases. Since it may be self limiting, in children splenectomy is rarely necessary.

(7) *Secondary thrombocytopenia.*—Treatment consists of specific measures for relief of causative disorder and steroid therapy. Steroids have a non-specific action lessening the capillary fragility. In drug induced cases the withdrawal of the particular drug is necessary in addition.

SURGERY

Even minor surgery should be avoided unless absolutely essential. Dental extraction should be done as an in patient under general anaesthesia after pre-operative blood transfusions. Adequate local coagulant plugging is essential. In children prophylactic dental hygiene is absolutely essential.

Surgery.—Arterial ligature may be necessary in some vascular disorders.

Parenteral injections.

Even more than normal children, haemophilic and children with other bleeding diathesis need immunization against poliomyelitis, diphtheria, whooping cough and small pox.

These can be undertaken provided sufficient digital pressure is maintained at the site of injection for about 5 minutes.

SOCIAL ASPECTS

Problems arise in these patients regarding schooling, occupation, rehousing, transport, convalescence, etc.

Schooling.—Each case has to be studied and considered on merits. Child can go to school provided the teacher and school medical officers are well informed and are willing to co-operate. Additional home teaching can be done to overcome frequent absences.

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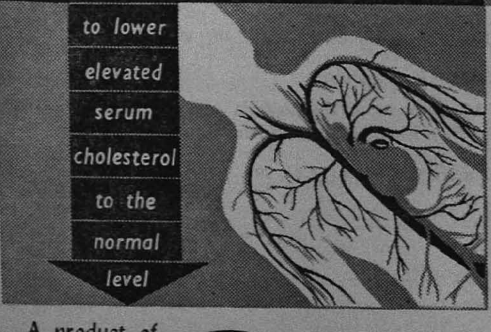

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Multiple Myeloma

Dr. E. MEENAKSHISUNDARAM, M.B.B.S.*

INTRODUCTION

Multiple Myeloma is also known as Plasmocytoma, Myelomatosis, Haematogenous Myeloma and Kahler's disease.

Multiple myeloma is a neoplastic disease occurring in multiple foci, caused by proliferation of abnormal plasma cells in the bone marrow.

Multiple myeloma, though a rare disease, is not uncommon. The incidence is increasing in recent years because of greater awareness of the disease, more number of cases being subjected to sternal aspiration and electrophoretic analysis and also more patients in the fifth and sixth decade are getting admitted into the wards.

HISTORY

In 1845, Sir James Watson while examining a patient found in his urine a peculiar substance which solidified on cooling. Being curious as to the nature of this substance, Dr. Watson sent samples of urine to Henry Bence Jones. After a considerable study, Bence Jones found that this unusual urinary substance precipitated on heating, cleared on boiling and returned to solid state on cooling. He also noted at necropsy that the "bony structure of the ribs was cut with the greatest ease, and that the bodies of the vertebrae were capable of being sliced off with a knife. This protein is now known as Bence Jones protein and the disease with which it is commonly associated, multiple myeloma. In 1846, Dalrymple suspected the malignant nature of the disease. In 1889 Kahler ascribed the four cardinal findings

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to this disease, *i.e.* (1) bone pain, (2) deformation and abnormal fragility of bone, (3) cachexia and (4) the presence of Bence Jones protein-uria. In 1899, Ellinger first noted the inter relationship of hypoproteinemia and mutiple tumours of bone. Longsworth, Shedlovsky and MacInnes were the first to make electrophoretic studies in cases of multiple myeloma and pointed out the existence of unusual patterns in this condition.

INCIDENCE

Adams and others working in Rochester Hospital, New York, reported the incidence of multiple myeloma as 0·02% of all hospital admissions and 0·66% of all malignancies.

AETIOLOGY

The etiology of multiple myeloma is not known. But the condition is comparable with lymphatic leukaemia in which the bone marrow throughout the body is involved. Since the lymph nodes as well as the bones are involved in multiple myeloma, some workers consider that multiple myeloma should not be regarded as a bone tumour but as a disease of the blood forming organs like leukaemia. It may start in the bone and spread later to lymph nodes or *vice versa*. It may remain confined to lymph nodes or bones. The myeloma cell is now generally believed to be a derivative of the reticulum cell.

Although multiple myeloma is comparable to leukaemia, it differs from it:—

(1) in the sharper localisation of the neoplasia,

(2) the frequent absence of enlargement of spleen or lymph glands,

(3) the much smaller tendency for the abnormal cells to enter the blood stream and

(4) the frequent appearance of Bence Jones protein in the unine.

Intermediate form occurs with features of both the diseases.

AGE

More frequent between 50 and 70 years; commonly at the age of 55 years; rare before 40. Cases of children having multiple myeloma have been reported. But they should all be regarded as medical curiosities.

Sex.—Males are more affected than the females in the ratio of 3 : 2.

Race.—Negroes are more affected.

PATHOLOGY

The tumour is first recognised as a widespread hyperplasia of the marrow cavity of cancellous bones such as spines, ribs, sternum, skull, pelvis, etc. Localised multiple accumulations of tumour cells occur within this generalised proliferative process.

The tumour is multiple, small—may be a few mm. in diameter or occasionally reach a diameter as great as 5 cm.—and as a rule soft and haemorrhagic. Degenerative softening and cyst formation are of frequent occurrence. Occasionally there may be a single tumour.

Multiple foci of tumour cells erode the bone, expand the cortex and destroy it causing bone pain, rarefaction of the bones, deformities and fractures. The bone becomes so soft that it can be readily cut with a knife. Occasionally, bulky tumour masses extend through the bone into the soft tissues.

Metastases are not characteristic of this disease, but they may occur in the liver, spleen, kidney, lung and ovary. There may be extradural pressure on the cord with the production of sensory symptoms which sometimes leads to the mistaken diagnosis of pernicious anaemia. The spleen may be moderately enlarged.

The myeloma cell may be a small cell with scant cytoplasm and an eccentrically placed nucleus which closely resembles a

normal plasma cell. Or, it may be a large anaplastic cell with abundant, vacuolated cytoplasm and an eccentric nucleus with one or more nucleoli.

ABNORMAL PLASMA PROTEINS

Myeloma cells are responsible for the abnormal proteins that are found in the serum, urine and tissues.

Serum.—In most of the cases, total protein is increased and globulin is also increased even as much as 8 g% (normal 2 g%) The formol gel reaction is positive. Even though the globulin is increased, the normal gamma globulin fraction is decreased, due to the depression of the synthesis. This leads to a deficiency of circulating antibodies. In some patients with multiple myeloma, inadequacy of the immunological response may be an important factor in the recurrent attacks of pyogenic infections and bacterial pneumonias which are so common in this disease.

The use of electrophoresis of plasma has shown that each patient with multiple myeloma would appear to produce his own individual "myeloma protein", which remains unchanged. This myelous protein or "M" protein moves between the gamma globulin region and the beta globulin. When this "M" protein is found in the serum, then the disease is known as "M" protein myeloma. Apart from this "M" protein, there are other abnormal proteins which move in the region of gamma globulin or beta globulin or alpha 2 globulin. The disease is then known as gamma protein myeloma, or beta protein myeloma or alpha protein myeloma respectively.

"M" protein myeloma is the commonest. Beta and gamma protein myeloma come next in frequency. Alpha protein myeloma is rare. A case has been reported from Dr. R. Subramaniam's Unit, in which the alpha protein was increased in the serum electrophoresis.

Another case has been reported from Dr. Annamalai's Unit, in which the abnormal protein was found away from the gamma globulin along with other abnormal proteins. This is an

unique case because so far only three such cases with this abnormal protein have been reported and from India, this is the first case. Dr. Ninan Verghese has worked on this problem and published an article in the " Lancet ".

Another abnormal protein that may be found in the serum of the patients with multiple myeloma is cryo-globulin. The cryoglobulins consist of a group of serum proteins which have the common physical property of precipitating on exposure to cold and redissolving when returned to body temperature. In about 50% of the patients with cryoglobulinemia, multiple myeloma is found. In the other 50%, it is essential cryoglobulinemia.

Urine.—The urinary proteins are distinct and different from those found in the plasma. Each patient produces his own unique urinary proteins.

Proteinuria characteristic of myeloma can be detected by Bence Jones heat coagulation test. Bence Jones protein · is precipitated as a cloud when the acidified urine is heated to 55°C, redissolves at 85°C, but reappears on cooling. If albumin is also present, filter the urine specimen while boiling, thus removing the coagulated albumin from solution and leaving the Bence Jones protein in the filtrate.

Early in the course of the disease, the appearance of Bence Jones bodies in the urine may be intermittent and may become constant only late in the course of the disease. Its occurrence may be continuous or periodic. Most (about 65% to 75%) but not all cases of multiple myeloma will show a positive Bence Jones Protein from a trace to large amount.

Aside from these variables, the behaviour of the protein depends on many circumstances such as the concentration of Bence Jones Protein, the pH of urine, the concentration of urinary electrolytes, urea and albumin.

The occurrence of Bence Jones proteinuria has been reported in diseases other than multiple myeloma such as metastatic

carcinoma, lymphatic leukaemia, myelogenous leukaemia, senile osteomalacia, fibrocystic disease, multiple fractures of bone, inactive tuberculosis, etc.

Bence Jones protein precipitates in the tubules, producing acidophylic casts. These cause obstruction and subsequent atrophy. For this reason, many cases terminate with renal failure.

The presence of Bence Jones protein and other urinary proteins which are pathognomonic of multiple myeloma, but which do not have heat coagulability properties recorded by Bence Jones, may also be determined by electrophoresis of the urine.

Here also abnormal proteins (such as alpha, beta, gamma and "M" proteins) have been identified in the electrophoresis of the urine. But the abnormal proteins found in the urine are not the same as that found in the serum.

Tissues.—Para amyloid or para protein, another abnormal protein product of the myeloma cell, has the distribution of *primary* systemic amyloidosis in the myocardium, gastro intestinal tract including tongue, skeletal muscle, tendon, bursae, joint capsules, peripheral nerves, skin and larynx.

The para amyloidosis of myeloma less commonly involves the liver, spleen, and the kidneys than does the *secondary* amyloidosis of chronic suppuration. Again in sharp distinction to secondary amyloidosis, the glycoprotein deposit in myeloma does not stain well with metachromatic stains.

Affinity for congo red is also quite variable, making this test of little diagnostic value in detecting the para amyloidosis.

Amyloid deposits may occur in the tumours themselves, where they often are surrounded by giant cells.

CLINICAL FEATURES

Multiple myeloma may be *asymptomatic* and this asymptomatic stage has been observed upto three years before the onset of signs and symptoms of a typical case.

Weight loss is slightly early in the course of multiple myeloma. As the disease progresses and particularly if uremia develop, the loss of weight becomes pronounced.

Gastro-intestinal symptoms.—Anorexia, nausea, vomiting, diarrhoea and constipation may be present but not distressing unless azotemia develops.

Fever is a common accompaniment of the disease. It is usually low grade, remittent and resembles the febrile course sometimes seen in tuberculosis. Terminally the fever may go high and this terminal hyperpyrexia cannot always be explained on the basis of pneumonia or other existing infection. Its etiology remains obscure.

Pallor.—The pallor is many times associated with a peculiar, dusky, sallow colouration which cannot be attributed to anaemia *per se*. This peculiar appearance of the skin is not distinctive of myeloma, for it is often seen in other malignant states.

Anaemia although a fairly constant finding, is seldom extreme. The anaemia is in part due to replacement of the marrow by the tumour cells and in part due to an increased rate of RBC destruction. Haemopoietic tissue replacement may also be manifested by pancytopenia. The anaemia becomes aggravated in the terminal stages. It is usually normocytic and normo-chromic in type. Macrocytic type may also be found.

ABNORMAL BLEEDING

There is thrombocytopenic bleeding secondary to bone marrow replacement. In addition, there is interference with blood coagulation due to low concentration of prothrombin and the accelerator globulins as well as to interference with conversion of fibrinogen to fibrin. Cryoglobulinemia may lead to peripheral capillary thrombosis and haemorrhage due to endothelial membrane damage. Haemorrhage from the nose, gums, lungs, gastro-intestinal tract and skin has been reported.

Pain.—The pain is of two types: (1) that due to bone pain *per se* and (2) that due to fracture of bone.

Bone pain is insidious in onset, sometimes remittent in type, resembling "rheumatic pains". These pains usually progress and terminate in agonizing spasms. The pain may be localised in the thorax, abdominal or lumbar regions and sometimes in the neighbourhood of joints. The spine, ribs and sternum may be tender on percussion. The pain may also be widespread and fleeting, as there is frequently a generalised demineralisation of the skeleton.

Fracture pain.—When there is a pathological fracture incurred by a sudden movement or the lifting of a heavy object, the pain is acute and sudden in onset. The pain is frequently limited to the back or chest and may be of such severity as to cause the patient to cry out or fall to the floor.

In 60% of all cases of multiple myeloma, spontaneous fracture occurs. In no other type of bone tumour does pathological fracture occur so frequently. The fracture occurs in one or more ribs, clavicle, sternum and later in the long bones, proximal ends of humerus and femur.

Deformities.—Progressive kyphosis or angular curvature of the spine due to collapse of the vertebral bodies with loss of total weight follows:

Poor dentition.—A pronounced carious condition or a complete absence of teeth may be the commonest clinical finding. It should be realised that the majority of even normal people in this age group have either total or partial loss of teeth. However, it seems outside the realm of chance that practically all cases of multiple myeloma should show this, unless there were some other factors which predisposed to dental softening. The purely speculative hypothesis is offered that the same process which so commonly leads to skeletal osteoporosis also affects the teeth and leads to their premature decay and removal.

Palpable liver and spleen have been reported.

Palpable tumours.—Rarely the presenting complaint was a palpable tumour attached to the bone. In general, these tumours are soft, frequently pulsatile masses and were attached to the sternum, skull, ribs, clavicle or jaw.

Renal insufficiency.—Various theories as to how Bence Jones proteinuria interferes with renal function have been postulated.

(1) Some believe that there is a " toxic nephritis caused by elaboration of Bence Jones protein ".

(2) Others have postulated that glomerular damage results from filtration of Bence Jones bodies.

(3) Still others believe that damage results from the ischaemia caused by large amounts of abnormal protein circulating in the glomerular tuft.

(4) Actual tubular obstruction by protein casts associated with degeneration of the renal unit has been considered to be of primary importance.

When once Bence Jones protein is detected in the urine the development of renal insufficiency has begun. But even here in about 55% of patients with Bence Jones proteinuria, evidence of significant compromise of renal function has been found.

Nephritis without hypertension is fairly common in this disease.

So the true pathogenesis of renal insufficiency in this condition remains a mystery.

Para amyloidosis.—The signs and symptoms are related to the sites of deposition. Intractable cardiac decompensation may result from the deposition of the glycoprotein in the myocardium. Involvement of the alimentary tract, from the tongue (macroglossia) to the rectum may lead to dysphagia, malabsorption syndrome, diarrhoea and gastro-intestinal bleeding. Para-amyloid deposition around and within the peripheral nerves may lead to paraesthesia, anaesthesia, motor weakness of the extremities and

other physical signs compatible with peripheral neuropathy. Accumulation of material around the joint capsules may produce pain, and limitation of movement.

INVESTIGATIONS

(1) R.B.C. count : HB%.

(2) Total W.B.C. count.

(3) Blood smear :

Even in the blood smears the red cells are seen to be piled upon one another due to the increased tendency to rouleaux formation. Besides this, there may be a peculiar bright blue colouration of the peripheral blood smear (Wright's stain). This phenomenon has been attributed to the elevated plasma proteins. Adams and others emphasize the importance of these observations which are sometimes of help in the diagnosis of multiple myeloma.

In a few instances plasma cells may enter the blood stream in larger or smaller numbers and it is probable that they can be found if a careful search is made. Sometimes they are present in such large numbers as to justify a diagnosis of plasma cell leukaemia.

(4) E.S.R.

(5) *Serum calcium and phosphorus levels.*—The serum calcium is usually normal. The combination of immobilization and active bone destruction may lead to hypercalcinuria and hypercalcemia characterised by nausea, vomiting, constipation and lethargy. The elevation of calcium within the renal tubules may lead to the deposition of calcium salts, which, perhaps together with a direct toxic effect of the elevated calcium concentration on the tubular cells, may lead to uraemia.

The metastatic calcification while by no means constant, has been frequently observed in the kidney, lung, stomach, myocardium and uterine mucosa.

The serum phosphorus is usually normal. But where renal insufficiency complicates multiple myeloma, the serum phosphorus is bound to be high and may rise as the kidney condition becomes worse.

The parathyroids are not enlarged in multiple myeloma.

(6) *Phosphatase.*—The acid phosphatase is normal and the alkaline phosphatase is normal or slightly elevated in multiple myeloma.

(7) *Serum uric acid level.*—In a number of neoplastic diseases such as polycythemia, leukaemias, and lymphomas—as well as multiple myeloma, there is an increased rate of purine metabolism due to the formation and degradation of the tumour cell nucleic acid. Uric acid is the final step in purine catabolism and on occasion the concentration of uric acid is significantly increased in the serum of patients with these diseases. The hyperuricemia rarely may lead to secondary gout. Urate crystals may be deposited in the kidneys, leading to obstructive uropathy.

(8) Formol gel reaction.

(9) Electrophoretic pattern of plasma proteins.

URINE

1. Bence Jones Protein.
2. Electrophoretic pattern of urine proteins.

Bone Marrow :

1. Bone marrow—Sternal puncture.
2. Biopsy of a portion of affected bone.

X-ray of various bones to observe characteristic features, any fractures and deformities.

In X-rays, the marrow tumours are found mainly in the spine, ribs, sternum and skull. They are seen as clean-cut

elliptical or circular areas of complete translucence, set closely together, and varying from 1 mm. to 5 cm. in diameter. The large tumours may expand the cortex of the bone affected. There is a good deal of generalised osteoporosis throughout the affected bones. The spine shows collapse of the bodies of one or more vertebrae. The skull is not thickened. Spontaneous fractures especially in the ribs are very common.

There are few cases of multiple myeloma which never exhibit X-ray changes of any type.

Differential diagnosis.—The commonest diseases which are apt to be confused with multiple myeloma are metastatic carcinoma, arthritis, anaemia, chronic nephritis, hyperparathyroidism, Paget's disease, ruptured nucleus pulposus, osteomyelitis, leukaemia and tuberculosis. It will be seen that the above diseases fall into four main groups:

1. Disease of bones.
2. The anaemic states.
3. Renal disease.
4. Certain febrile illnesses.

The differential diagnosis of disease of the bones is aided by a careful study of serum calcium and inorganic phosphorus levels and a determination of the alkaline and acid phosphatase activity. When this information is added to careful roentgenographic interpretation much confusion may be averted.

The elucidation of unexplained anaemia, whether it be macrocytic, normocytic or microcytic, is aided by the examination of a suitable bone marrow preparation taken from sternum.

The frequent occurrence of Bence Jones proteinuria, the low incidence of hypertension and the inability to demonstrate renal disturbance by roentgenographic means all contribute to distinguish the renal insufficiency of multiple myeloma from other renal disorders.

Various febrile illnesses may be confused with multiple myeloma unless it is realised that fever is a common accompaniment of the multiple myeloma.

Diseases.	Calcium.	Phosphorus.	Acid Phosphatase.	Alkaline Phosphatase.
1. Hyperparathyroidism.	Elevated.	Depressed.	Normal.	Elevated.
2. Paget's Disease.	Normal.	Normal.	Normal.	Elevated.
3. Metastatic Carcinoma to bones.	Elevated frequently.	Normal.	Elevated in metastasizing carcinoma of prostate.	Elevated.
4. Multiple Myeloma.	Elevated frequently.	Normal.	Normal.	Slightly elevated.

PROGNOSIS

The disease, although not highly malignant in the sense of a rapid termination, is invariably fatal.

Death often occurs within 6 months of the onset of symptoms, but occasionally a patient survives for two years or more.

Broncho-pneumonia, cachexia or compression paraplegia with ascending pyelonephritis are the usual terminal events.

The vast majority of patients pursue a continuous downhill course from the onset of symptoms to the time of death. In the series of 61 cases studied by Adams and others, it has been found that the longest survival time was five years and the shortest one month.

TREATMENT

1. *General Supportive Treatment.*—Early ambulation of the patient who has become bed ridden because of pain and discomfort. This measure is emphasized because decalcification of the skeleton proceeds at an accelerated rate on immobilization. This, coupled with tumour erosion of the bone, may lead to hypercalcemia with renal damage and uraemia.

2. *Hydration* in the presence of hypercalcemia and hyperuricemia to prevent damage to the kidneys.

3. *Blood Transfusion* to combat anaemia.

4. Prednisone (5 to 10 mg. t.d.s.) to decrease the rate of R. B. C. destruction.

Specific antitumour treatment consists of chemotherapy, radiotherapy and surgery.

1. *Chemotherapy.*—Urethane (Ethyl carbamate).

It produces significant objective and subjective remissions in 30 to 40% of patients.

It is best given as a 10 to 20% flavoured solution at a dosage of 2 to 4 gm. daily for a minimum of 4 to 6 weeks. The liquid is preferred to enteric coated capsules because the capsules are frequently inadequately digested and absorption of the urethane from the intestinal tract is incomplete.

The drug is given in maximal tolerated doses, gastrointestinal symptoms often constituting a limiting factor. Complete blood counts should be made at weekly intervals and therapy discontinued when the leucocyte count is depressed below 3000 or platelets are below 80,000. An effort should be made to produce evidence of haematopoietic depression before considering the drug ineffective since this is the only reliable indication that a pharmaco-dynamic effect has been achieved.

When a remission is produced by urethane, therapy may be discontinued. Exacerbation of the disease should be again treated with urethane.

2. *Corticotrophin or steroids* may give temporary relief of bone pain and other indications of brief remission.

3. *Stilbamidine and hydroxy-stilbamidine.*—Favourable responses are too irregular and toxicity is too great to recommend use of these compounds.

Radio Therapy.—It is valuable in alleviating severe localised bone pain and in reducing the size of the tumours. It is used in combination with surgical decompression of an epidural tumour mass impinging on the spinal cord.

Surgery.—When the symptoms and signs point to the compression of the spinal cord, surgery is indicated. Laminectomy reveals a grey or reddish-grey extradural mass either pushing the cord backward or encircling it. Removal of the mass decompresses the cord and is followed by improvement. Deep X-ray therapy and wearing of the spinal brace are advised after laminectomy.

SOLITARY MYELOMA

(Solitary Plasmocytoma). Rarely, myeloma may be apparently localised to be a single focus such as, the rib, pelvic bones, spine and mandible. It manifests as a distinctive bone lesion in the intramedullary cavity. In the majority of cases, the usual clinical picture of multiple myeloma ultimately develops, usually after many years.

In multiple myeloma, aspiration of the bone marrow, wherever it is made, usually reveals myeloma cells in the absence of X-ray changes in the bones. It is not so in solitary myeloma.

In solitary myeloma, myeloma proteins and Bence Jones proteins are absent from the serum and urine, usually but not always.

Vigorous X-ray therapy or surgical resection may be followed by prolonged remission or occasionally apparent cures.

EXTRA MEDULLARY PLASMOCYTOMAS

Their greatest frequency is in the upper respiratory tract including the tonsils. They have also been found in the gastrointestinal tract, pancreas, pleura, kidney and skin.

They may be histologically indistinguishable from myeloma cells of the bone marrow. The protein abnormalities in the serum are identical with those observed in skeletal myeloma. But Bence Jones proteinura has not been reported in the absence of bone involvement.

The entity of extra-medullary plasmocytoma is unquestioned. But the diagnosis is made more frequently than warranted. It is to be emphasized that inflammatory granulomas may be rich in plasma cells. This must be considered whenever a diagnosis of extra medullary plasmocytoma is suggested.

The tumours may invade locally and metastazise to lymph nodes and bones. Death may result from infiltration of kidneys, liver or heart or the clinical picture of classic multiple myeloma may emerge.

The therapy of extramedullary plasmocytomas is by radiotherapy when it is localised and by urethane when it is disseminated. There is an impression that localised extramedullary plasmocytomas can be effectively controlled by irradiation.

CONCLUSIONS

The incidence of plasma cell tumour is greater than had been previously suspected. It is the responsibility of the clinician to recognise the cardinal signs and symptoms of this condition and to request the proper laboratory studies which may corroborate such a diagnosis. It is self-evident that unless the existence of the disease is suspected by the clinician, its "accidental" laboratory discovery will be rare. In our present state of knowledge, very little of a specific nature can be done for these patients. However, in the interest of making earlier and more accurate diagnosis than in the past, the following suggestions are made:

1. In any case in which either hyperproteinemia or hyperglobulinemia is discovered, careful electrophoretic studies be carried out.

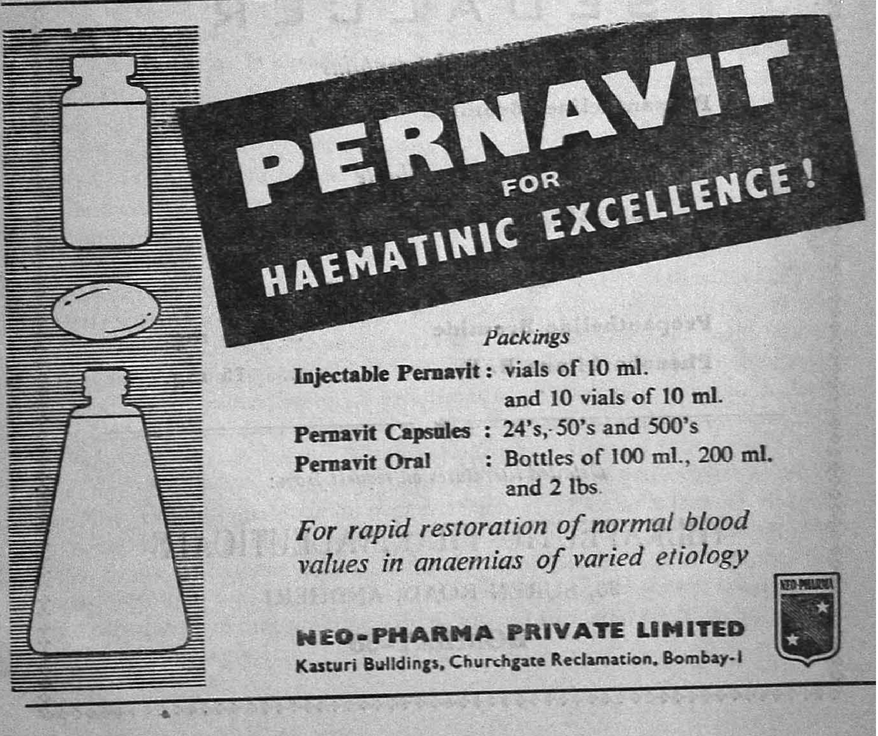
2. More attention be paid to the occurrence of rouleaux formations of R. B. Cs. in the peripheral blood and to the bright blue colouration of the blood smear, and that a satisfactory cause for their presence be found.

3. Any case of unexplained anaemia, particularly in the older age groups be given the benefit of a sternal aspiration and or sternal biopsy.

4. More energetic and careful searches be made for Bence Jones protein in all urine specimens which contain protein.

5. The level of serum calcium, inorganic phosphorus, alkaline and acid phosphates be determined in suspicious cases.

6. Careful analysis be made of all roentgenographic osteolytic processes and more attention be paid to the occurrence of osteoporosis.



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TRANSCRIPT OF THE ADDRESS

on 30th December 1960

AT

MADRAS MEDICAL ASSOCIATION ANNUAL DINNER

BY

S. BALASUBRAMANIAM,

Dean, Stanley Medical College.

I consider it a singular and unique honour and privilege to be asked to fill in the role that I am performing tonight. I am conscious of my inadequacy for the task. It has all along been given to the stalwarts in our Service, men of excellent attainments and such like, to be invited to perform this task. I am neither vain nor foolish enough to suppose that I have any pretensions to such high distinctions. At best, the only claim, if any, I can make for myself is that I have played the game in the correct spirit to the best of my understanding for the last thirty years and more in all the various situations pertaining to our Service which I happened to occupy. I am just a common man but then the common man is coming into his own in our present Socialistic State of Society. In fact the common man is often invoked for diverse purposes and so perhaps it is not inappropriate that you should have thought fit to exalt the common man for your Chief Guest to night. It has happened that I have filled in all the different roles that one in our Service may be called upon to occupy. From that of an Assistant Surgeon, Assistant District Medical Officer in several districts for the first eight years since 1929, to the Madras and Stanley Medical Colleges as Assistant in Surgery for the next five years, back again to the Districts for the next fifteen after obtaining my Post-graduate qualification over ten years of which as a District Surgeon in some big hospitals in spite of enquiries to come back to Teaching, it has fallen to my lot to come again to the Medical Colleges in the capacity of Dean. So then I could claim that I have had opportunities of contact with medical profession and practice, education and administration during the last 31 years of my service.

The Subject of my Talk to night - I am rather nervous about calling it an Oration, shall be "Random Reflections on ourselves - Last Thirty Years". Lest I may be guilty of an inadvertent omission, I shall even now at the outset refer to one or two things. I would consider it as my paramount duty to pay Homage and my humble tribute to the glorious traditions of our Madras Medical Service since its inception. I believe I am not making any exaggerated claim about our Service which has apart from its commendable work in the field of Scientific Medicine and Health of our people in the decades past produced such great and outstanding men of Excellence, Character, Service and Devotion to duty as our Respected Vice-Chancellor Sir A. L. Mudaliar - who is still doing yeomen service not only to our State, our Country - nay the whole World; Dr. S. Rangachari whose statue in the General Hospital is a perpetual reminder to us all and medical students of successive generation for all that is best in our profession in all climes and countries and for all times; Dr. Guruswami Mudaliar whose name is one to conjure with, a lingering memory in the minds of thousands and thousands of his students and equally so of his patients. I could go on multiplying examples of such great personalities but I shall of necessity refrain from doing so for the simple reason that the time at my disposal should be entirely spent in such recapitulations without being able to exhaust them. Next point that I would refer to is this that we are at the moment extremely fortunate in having as the Minister in charge of Health a person of the eminence of Sri Manickavelu Naicker who has at all times evinced a keen interest in understanding the various problems of our Service, of the health of our people and has taken great pains to lose no opportunity in advancing their cause. May I next convey our grateful thanks for his kind presence here to our popular and learned Minister for Finance who in addition to husbanding the Finances of the State is ever ready to loosen the purse strings for all good causes! and what good cause can there be more than the Health of our People?

Tribute to Madras Medical Service.

Hon'ble Minister.

Gentlemen, our Service, the Madras Medical Service, is something more than other Governmental Services. We belong to a learned profession. One who practises a profession albeit a learned one contracts certain responsibilities which do not go with other avocations. The fundamental characteristic of a profession is that its votaries do not equate their work in terms of any remuneration. Though one even in a profession has to earn his livelihood like any other, the dominant motivating force in the performance of his work has to be the good of his

Learned Profession.

fellowman, fellow sufferer and the professional satisfaction one gets out of its correct practice. But then Bernard Shaw would not have it thus. Most of you should have read his play 'Doctor's Dilemma' particularly its preface. I shall quote a few of his more trenchant comments on our profession, if only to provoke us to a righteous indignation to falsify his heresies by our example. Here goes :—

Shaw and Medical Profession.

"The more appalling the mutilation the more the mutilator is paid. He who corrects the ingrowing toe nail gets a few shillings; he who cuts your inside out receives hundreds of guineas except when he does it to a poor person for practice.

The process of metaphorically bleeding the rich man is performed not only metaphorically but literally every day by Surgeons who are quite as honest as most of us.

The medical profession has not a high character. It has an infamous character.

As to the honour and conscience of Doctors they have as much as any other class of men ; no more and no less.

It is simply unscientific to allege that Doctors do not under existing circumstances perform unnecessary operations and manufacture and prolong lucrative illness.

The medical profession is a conspiracy against the laity to hide its own shortcomings. It cannot too often be repeated that an operation once performed nobody can ever prove that it was unnecessary. Operation is therefore the safe side for the Surgeon as well as the lucrative side. They exploit human folly, vanity and fear of death as ruthlessly as their own health, strength and patience are exploited by selfish hypochondriacs".

This may give us the impression that Shaw suffers from some kind of strong antipathy to Doctors as a class and uses his literary artistry in maligning them. But that this is not so will be seen from passages in this preface itself :—

"It has been my luck to have Doctors among my friends for nearly 40 years past — all perfectly aware of my freedom from the usual credulity as to the meticulous powers and knowledge attributed to them.

The true Doctor is inspired by a hatred of ill-health".

So then the conclusion is that Shaw believed in what he wrote. If we are to silence such Shawian criticism, we should all raise our practice to equal

our profession that elimination of disease is our goal, resulting in our own self-elimination as a class. If it appears to Shaw and his like that doctors can't but have a vested interest in disease - as no disease means no loving for the Doctor - we claim that it is a false appearance however logical it may be. We shall ever strive to maintain this dictum by our high code of ethics, ideals and selfless service in the years and decades to come in the cause of the ailing public. So then our responsibilities as medical men and again as Members of the Madras Medical Service are really great. When one thinks of the extraordinary hold the Service had on young medical graduates of our time and the conditions now prevailing in regard to recruitment to Service, one can't but be sad. If 200 vacancies are now offered for recruitment, about 100 apply. Even at the Service Commission interview the vast majority of them on questioning could not hide their feeling that they may not join if they happen to be posted anywhere except Madras or perhaps in some bigger towns. The factors that contribute to the creation of this damaging situation need to be carefully considered. In the first place the Servicc since the dawn of Independence is growing out of all proportion; not the normal healthy growth but a sudden uneven and uninhibited growth as if from some endocrine disorder. Secondly unless the Service evolves through the decades in a normal healthy fashion retirements being replaced year after year, expanding steadily and according to plan, giving opportunities for higher training in the country or abroad; the regular annual recruitment on an acceptable sound basis; unless these are done, thc morale and strength of the Service are bound to suffer. And this has happened to Ours. Thirdly the post-war effects on the minds and morals of people in all parts of the World have undergone a certain change, not quite desirable. And we of this part and this Service perhaps also share in it. Fourthly, the Service has been allowed to expand to a degree which could not be met adequately by the supply of Graduates from our Colleges though their numbers have reached several times over. Having said all this, I do not consider that they are a full answer. The young men and women who passed out of our Medical Colleges in recent years exhibit curious and ununderstandable attitude to Life. They all seem to have developed some type of Phobia for going out and working in the countryside. They seem to suffer from a lack of purpose and seem totally disorientated as to what they should do in life. It is alright for the bright students at the lowest, those who had the capacity to go through the medical curriculum in the minimum regulation time to aspire to continue post-graduate study and take a qualification before embarking on their career. But this can't and does not apply to the bulk of them. They should be prepared to enter medical practice either on their own or through our Service.

Madras Medical Service.

Paucity and its causes.

Young Graduates' Vacillation.

I believe that this unhealthy resistance to entry into Service stems from a proper lack of motivation and general liberal education during their years of College. How this could be met during the formative years of the student while still at College is a matter for consideration, and adoption of suitable measures. I believe that it is not merely the scales of pay that influence the would-be entrants to Service. The unexpressed desire in most of them, if I could correctly read their minds is opportunity for professional satisfaction. They seem to believe wrongly that it could be possible only in big hospitals in the Cities. Conditions of living, accessibility, remoteness of the locality, all these may have something to do with the paucity of recruitment. The student should be made to know that all these have undergone quite a rapid transformation in several parts of the countryside under the Community Development Programme. Sufficient and correct information should also be spread about the Primary Health Centre, the new type of Medical Institution started under the Plan Schemes. Primary Health Centre is a red-rag to the young medico. It should therefore be made clear to him that his impression is erroneous. Most of the Primary Health Centres are now housed in their new buildings and quarters for Medical Officers and Staff are under construction in most places. Each centre has a motor vehicle for tour in the surrounding villages; an adequate staff—Medical and Public Health-work under the Medical Officer. Essential equipments like microscope, sterilizer, operating table etc., are all provided. The opportunities for real service more than anything else are really great for any young man with some initiative and resource and a sense of duty towards his countrymen. So then my conclusion is that the medical student if properly motivated towards the Service which he owes his countrymen 85% of whom live in villages, if also properly informed about the conditions now obtaining in the various parts of the State concurrently with efforts to improve them, the problem of recruitment to our Service will satisfactorily be solved.

I believe that the present state of our Service is largely due to historical forces and factors arising from rather too rapid development uuder the Plan Schemes, following an earlier period of hybernation and inaction. The medical facilities provided during the British regime were far too inadequate. medical relief of the people of this country was not the main concern of the British Administration in India. So, the administration was content with doing the minimum that a given situation demanded for the peace of its own mind and incidentally giving an impression that the welfare of the people is at the heart of the Administrators. So, the neglect of centuries—may not be active but it is still a neglect—has to be repaired; and in the attempts to repair the damage by the various Plan Schemes the Service in the State is now, more particularly on its Health side, unable to deliver the goods as required. But I believe it is only

Neglect of Centuries.

a passing phase and it will be possible very soon to restore our Service as an instrument of high efficiency for the medical needs of such a small State.

I shall next deal with our Hospitals, teaching and non-teaching and dispensaries. The primary duty of a State is to provide conditions of healthy living, such as adequate food, good water supply, housing, elementary sanitation etc. These have not been done during the British regime. This lack, a bad legacy of the past complicates our problems of providing adequate medical relief. Secondly the problem in our hospitals for the medical man is not just one of medicine but it is medicine plus poverty and ignorance. To the extent these latter are eliminated by our plan schemes the problem of medical relief will also become easier. It is in such a context that we have to deal with the problem of our hospitals.

Our Hospitals and Dispensaries-Expansion and Improvements.

There has been a very rapid expansion of medical relief during the last decade after Independence. New buildings, addition to existing buildings and considerable amount of equipment have been added to all these institutions by the initiative and unremitting drive and efforts of our Director of Medical Services. As I have already pointed out in such a sudden and rapid expansion the greatest difficulty has been to find young medical graduates to fill up all the sanctioned posts, and other auxilliary medical personnel to satisfy our needs. The great problem in regard to hospitals is in my view the enormous overcrowding in them.

Floor Patients.

If there is one measure of reform more important than any other in the improvement of our Hospitals that should be the abolition of floor patients. After admitting one as an in-patient in a Hospital I feel very strongly that it is against all canons of decency, courtesy, consideration and proper care and treatment to put him on the floor on a mat on the specious plea that he is no better in his own home. Once some arrangement is made which will make it possible to admit in a hospital patients no more than its sanctioned number and will not allow any more, this will atonce have the effect of removing in one sweep so many evils in our hospitals and improve inmeasurably the standards of medical treatment and nursing care. I have been making this plea during the last several years and on one occasion on an enquiry from the Director of Medical Services, I have given full details about the cost of additional buildings that may be necessary to accommodate on beds the patients on the floor in all the hospitals in our State. Costly buildings are unnecessary for the purpose. The present practice of five feet space between cots, negativing its value atonce by putting two patients in that space is wasteful and meaningless. The ward itself can be designed so as to provide only a two and a half and three feet between cots. I have given detailed specifications on the matter working out the cost at rates prevalent in 1956. Even at the present rates it will be possible to put up ward buildings at not more than rupees

thousand per patient. In other words, the cost of 100 bed ward should be limited to one lakh of rupees. An addition of Rs. 200/- per bed will be necessary for furniture and ward equipment. This of course does not include buildings for out-patients, stores, theatres, etc. as it is my plan only to provide the additional wards necessary for each hospital to take up all its floor patients as an annexe. Taking roughly that the number of patients on floor in all our hospitals in the entire State to be 5,000 the cost for provision of additional wards for them will be only Rs. 50 lakhs. The recurring cost on account of staff for this scheme can be estimated roughly to be of the order of 20 lakhs per year. No separate commitment is involved in regard to drugs and diet as these are already provided for these patients in the existing hospitals. I submit that this expenditure cannot be considered an extravagant figure in relation to the enormity of good that it will do. To argue that if you seek to provide more buildings the number of patients will correspondingly increase necessitating floor patients again is in my opinion a defeatist attitude, particularly in the context of our increasing tempo of public health measures to eradicate and control many of the communicable diseases. To postulate an increasing incidence of disease and demand on hospitals simultaneously with efforts to eradicate poverty and disease is to negate the very purpose and objects of planning. In any case that the evil will continue in spite of our efforts is no answer to meet the present evil.

Proposals for Floor Patients.

The problem in Out-patient Department as among the in-patients is the enormous over-crowding. Poverty and ignorance contribute a lot to this. The majority of patients as many as 80 to 85 per cent come for functional, trivial and minor ailments. The task of the medical graduate is to spot in this crowd the patient suffering from structural disease in any organ. As a corrective against errors which may arise because of the crowds of patients, it is essential to have a review Out-patient where all patients who have attended the department for more than 10 days continuously will be re-examined to see if they are suffering from anything more serious than what was disclosed at the first examination. Secondly the diagnostic facilities in the Out-patient could be improved so that the necessity for admission as in-patient for investigation could be minimised to some extent apart from improving the standard of diagnosis in the Out-patient itself. It would be advantageous to employ Technicians, so trained from among B.Sc. graduates.

Other Improvements.

Periodical visits of Doctors working in Primary Health Centres and smaller dispensaries to the District Head-Quarters Hospital may be arranged, say for a week once in a quarter.

In hospitals with two or only one doctor the strain of medico-legal work particularly drunkenness certificates is far too much. This requires to be solved in some satisfactory way.

Hospital Administration.

Administrations all over the world are targets of attack and satire. Administration, if I may say so, is something undefinable, say like Providence, and perhaps subject to the same type of vagaries. Any one interested in administration, even a medical administrator, should have read a recent publication, namely the *Parkinson's Law*. Though it is a humourous satire on administration and administrators, it contains a lot of truths which should be taken notice of. He avers that work expands so as to fill the time available for its completion. His first Law is that increase of Staff in any office is totally unrelated to any increase in work - in fact the work may have decreased - and raises at a flat rate of 5·8 per cent per annum. He supports it with his own facts which are incontrovertible. I can't say that this is true of medical department. Our Staff may have increased even at a higher annual rate but the increase is no more than or perhaps may even be less than what could be justified by the increase of work. His next thesis or Law seems more relevant to us, as it may be to many others. I refer to the chapter on methods of high finance wherein the "Law of triviality" is enunciated. This law laid down is, that the time and care bestowed by Committees on schemes of planning are inversely proportional to the respective cost of the scheme. This could be illustrated in several of our actions. To put through a minor work costing five thousand rupees will meet with several objections, long arguments, protracted delay, finding of funds, etc. etc., however important the work may be. Far and away easier apparently is to get a sanction for a new Block costing 20 lakhs or another costing 10. Similarly one may be able to get a grant for a costly apparatus costing several lakhs in much quicker time than common essential needs of any ward. For example it is none too easy to get microscopes. Questions are asked, so many of them, when was the old one bought, is the request for replacement or additional one, how was it carried on earlier, the number of examinations done per day on and on. So much so, that it often happens that the file becomes dead either because the concerned clerk is himself sick of it or the officer has been too worried to worry more. Similarly in matters of staff what any one considers to be absolutely essential - say a Stenographer for each of the Professors or District Officers - is far more difficult to obtain than creation of higher paid jobs. You may say "why should this be so?". Nobody is responsible. It is perhaps the same as saying "all of us are".

Parkinson.

These are matters which should require critical attention ; in principle, stated briefly small essential and primary needs like the provision of minimum diagnostic equipments, staff like Technicians and Stenographers should be

made available without much fuss on the recommendation of the Heads of Districts or other Independent Units, giving only a second place or perhaps even a third to costly schemes which in themselves may be good but whose benefit to the generality of people will be only relatively small. Avoidance of costly buildings should be talked a lot less and practised a little more !

Medical Education.

World Conference.

Deans' Conference.

I.A.A.M.E.

I shall now come to the question of Medical Education. This is at present a very important and controversial problem engaging the active attention of medical educationists throughout the world. Naturally we in India have also decided that it is too late for us not to make a beginning. Not a day passes when some celebrity or other deplores from some platform the fall of educational standards and medical education takes more than its due share in this criticism. There have been two World Conferences of Medical education during the last decade. Coming nearer home there was a conference of Deans and Principals of Medical Colleges of India at New Delhi in September last for considering the problems of medical education. On the suggestion of some members attending that conference an Indian Association for advancement of Medical Education was proposed and this was duly inaugurated by our Union Health Minister and Vice-Chancellor at Hyderabad earlier this month. The curious thing is that during the last decade and perhaps little more a large measure of improvement has been effected in the staff, equipment, and buildings of the existing colleges. To say, that at any rate in them, the standard has fallen down - it seems to be so - is rather understandable. In our days, that is, during the late 20s any member of the I.M.S. could teach any subject. Our Vice-Chancellor never fails to make this joke, in any of his speeches in Medical College, that the Professors in his time got promoted along with the student. In my own case, I had the same Professor teaching my class in Biology, later Hygiene, later still Surgery. Most of the college departments had only one assistant and a few had two. Not all Professors had a post-graduate qualification and none among the assistants. The Teacher - pupil ratio was not heard of. The buildings and laboratories were unpretentious and minimum. Still the graduates in Madras Medical College in those days were considered very good and efficient. He had proved himself so whether in service, civil or defence or in practice. As against this picture the colleges are now vastly strengthened. The Professors are all highly qualified and experienced in their respective branches. There are quite a few post-graduate qualified assistants. The number of teaching staff has increased. The hours of teaching tutorial and coaching classes have all been increased. To talk of lower standards along with this, if something very extraordinary, requires very careful thinking and analysis as to why it should be so, if it is true.

The Anomaly.

There is ofcourse the problem of the newer medical colleges. They are increasing in numbers very rapidly. In our own case, when I joined College there was just one, the Madras Medical College for the whole of the composite Madras State plus Mysore and Travancore. In the same area now we have 18. The number of colleges in all India has doubled during the last decade—the number now being about 59. Quite apart from the cost of a new medical college which is soaring higher and higher every day, the greatest difficulty is finding of required staff with the prescribed qualifications particularly in the pre-clinical subjects. This is a problem of some magnitude and can conceivably result in lowering of standards.

Causes - Lower Standards.

(1) Paucity of Staff.

Another factor tending towards this is that in order to meet the requirements of a country's need for more and more Doctors the number of admissions to medical colleges has progressively increased. The optimum annual intake in the Medical College is 75, but now many of the Colleges even those started very recently take 100 and more; some take as many as 150. This is obviously not very desirable though it is considered an easier and cheaper method of turning out a larger number of Doctors than starting of new medical colleges. If there be any substance in the fear that there is deterioration of standards in medical colleges it seems obvious that we should not embark upon opening of more till we consolidate the position of the present. It will be argued as to how then we are to meet the demand for more doctors to meet the needs of our villages. 3 new colleges and expansion of 3 old per year is kept on regularly till 1981, will then give a Doctor population ratio of 1 to 3805 as against 1 to 6118 in 1961. It is in this context that one has to consider whether we should not make better use of our available medical personnel, whether it will not be more in accord with the needs of our villages to train a category of people who may be called "*Health Assistants*" for rendering first aid and treating minor ailments and attending to health matters in a village under the supervision of a qualified medical graduate, the details of which could be worked out. This was a proposition made out by the Union Health Minister in his address at the opening of the I. A. A. M. E. at Hyderabad. The proposal is apparently so made in order to meet the criticism of constituted medical bodies against the introduction of a qualification in medicine lower than the M.B., B.S. In any event the possible advantage of starting a course for Health Assistants in villages will have to weigh against the disadvantages that may follow upon the starting fresh medical colleges without the availability of qualified personnel in adequate numbers, apart of course from the huge cost of a new medical college.

(2) Increased Annual Intake for more Doctors.

Health Assistants

I would now proceed to consider a few other important aspects of medical education.

Selection of Students.

This is a very important problem, probably a controversial one. We may confine ourselves only to scientific aspects of the matter. The entire difficulty arises from the fact that the available places in spite of the vast increase in the medical colleges is only about a third of the number of applicants. This does not mean ofcourse that all those who apply are fit for a medical career. It is said that medicine is not a career for average people and that one should try against the introduction of mediocrity into medical standards. But then it is impracticable in our country where reservations are necessary for the less advanced sections of the population. This course is an arduous one, perhaps the most arduous and long among the professional courses. It requires of its students continuous application and industry and endurance with at least a measure of intelligence. The easiest method to determine the number required from among the applicants is to select them in the order of their previous academic proformance. But, this is by no means infallible. To try to judge at the age of 15 whether the boy has shown evidence of possessing qualities and attributes of a good doctor is difficult. The selection of the top few, say the top 20 per cent or so is easy enough. Their academic performance is outstanding and provided they are physically fit they get easy acceptance. The boys in the bottom 30 per cent among the applicants who should never have wasted their time or the time of the Selection Committee, could also easily be disposed of by summary rejection. The trouble comes in the middle set forming 50 per cent applicants. No suitable device has yet been devised to ascertain if a boy could show evidence at an interview of his superiority over one who has scored higher marks. The assignment of some marks for extra curricular activities, say a maximum of 10, will be an easy matter which could be done. Barring that, the result of a short interview lasting not more than 5 mts. cannot in the very nature of it yield any reliable objective evidence of the merits of a candidate besides his academic performance.

The Medical College admission test as in America has been tried in a few Colleges and it is found that the results of such a test correspond closely to the Academic performance of the candidate in the earlier public examination. A third method of selection by observation of the applicants for a period as long as a week by Psychologists or Educationists is not reported to have yielded favourable records. So it appears that this matter requires further careful study in order that the best among the applicants may get admission with due regard to reservation for the less advanced sectors of the Community.

Cost.

The cost of starting a new Medical College is steadily rocketing up and is causing concern to authorities. Though we should learn by the experiences in the advanced countries of the West, it is necessary in a country like ours not to indulge in wholesale imitation but frame our requirements in a manner consistent both with efficiency and cost.

Curriculum.

A few words about the curriculum. It is generally agreed that the curriculum is becoming more and more crowded, leaving little time for the students to assimilate, much less to think. The main threat to the medical course as an instrument of modern liberal education is a very obvious one consisting in the enormous hypertrophy of detailed factual knowledge. This is the result of a vain search for comprehensiveness. The curriculum is punctuated by successive hurdles of formal university examinations, the first and last of which are found by the students to be ordeals of fearsome magnitude. The introduction of a compulsory

Intern Year.

intern year just after the final year examination has narrowed the wide gap between what should and what can be achieved before graduation. The endeavour thus by the introduction of the intern year is to make the medical course full practical, for practical skills can only be developed by practice under supervision and not by naked instruction. As general practice is becoming increasingly recognised as the greatest of the specialities a discipline attracting more medical graduates than any other it is obvious that the benefit of the intern year are really worth-while. On the other hand it is felt by many that an year of internship is too long a period as an addition to the already long medical course and that further it has not yielded fully the results expected of it. This again is a matter which requires further consideration.

Failure Rate-scope of Problem

The most important problem in medical education is increasing number of failures and the decreasing number of students who are able to complete the medical course within the minimum period prescribed by the Universities. The magnitude of the problem should atleast be roughly assessed before seeking remedies for it. I say this because there is a tendency to use damning superlatives in regard to this problem of failures. The number of students who discontinue this course on this account are indeed very few and the problem as such needs no consideration. The failures simply mean that the bulk of students-may be as high as sixty to seventy per cent–take between one and two years longer to complete. About 10 per cent of the students become chronic staying longer in the College, the record in this respect being 18 years. Obviously this last category have, if I may say so, trespassed into a medical college! They should not have been there; perhaps they should not have been admitted to any course of university education in their own interests. It happes to be in the national interest too. To submit a student,

in the guise of doing him good, to take the course in a medical college when he is totally unfit to cope with it, is a rank piece of cruelty. It is indeed tragic to meet them and try to do something to solve their problems as I have often to do.

In the case of the 60 to 65 per cent who take one to two years longer, I believe it should be possible to do something to shorten, if not eliminate this increased period by suitable alterations in the curricula methods of teaching etc. This question of failures is again intimately connected with the problem of selection of students for admission. To the extent this could be perfected the problem of failures will recede. As it happens now, the students constituting a class are not within an acceptable range of academic capacity. In a class of about 100, the top 20 take care of themselves given the opportunities for learning. The bottom 10 to 15 should not have been there. What remains requires to be properly handled to get the best out of them. In such an educational convoy, if I may use this term, the speed is that of the slowest ship. It is obviously important to avoid such a situation in the medical course. The student who is agreed by all teachers to be an idle misfit and who merely sets the final seal on his lack of endeavour by failing time and again to pass formal examinations should have found no place in a medical college. It seems often to be the impression that when one talks of failures one seems to think that this affects the annual output of doctors. This is not correct. For instance in older colleges like Stanley Medical College the number of annual admissions is 110. Obviously the output can be no more than 110 if all of them pass without halting anywhere. This ofcourse does not happen. Even so the number of graduates passing out of the College annually for the last several years is a few more or less than 110. The difference is only this much. Among the 100 who pass out - say in the current year's two examinations - there may be as few as 25 to 30 who joined the College in July 1956, the rest those who have joined a year or two earlier. If this is properly understood it will be seen that the problem is not so tragic as it might appear, when viewed from the national standpoint. It should not be understood that I am trying to belittle the evil effects of failure but one should not get panicky if a correct solution is to be sought. The factors which need attention in regard to this problem are rationalisation and relieving the congestion in the curriculum, improved methods of teaching paying special attention to the backward group, improvement or perhaps substantial alteration in the methods of assessment of the students' performance right through the course without having to rely entirely on the university examinations as the only test and an adequacy of teachers quantitative and qualitative. These are matters for consideration.

The subject is a vast and complicated one. I do not presume to offer my opinion on its various aspects. My only attempt is to pose them for consideration.

Improvements to Medical Colleges.

Time and again public men, men in authority, educationists have all given expression from public platforms as also in the Press that it is wasteful to sink lakhs and crores on gigantic buildings. None misses an opportunity to emphasise that it is the man and his tools that count in any venture. But curiously in actual practice buildings do absorb tons of money. Its justification appears often specious, but when it comes to staff particularly the lower ones as Technicians, Stenographers, etc., and day to day equipment there seems to be some type of administrative resistance. I referred to it already under Parkinson's Law. This will have to be reversed.

Buildings *vs.* Staff and Equipment.

Post - Graduate Medical Education.

This is important from mainly two points of view;

(1) Training of medical teachers to fill up the existing shortage as well as the need for the ever increasing number of newer medical colleges;

(2) For Research.

The first is more important from our point of view. This has been the subject for consideration of distinguished committees who I am sure would lay down the correct solution. It is worthy of consideration by the Director of Medical Services and the Government whether from among the passed out graduates in the State every year - there will be about 300 of them - 5 to 10 of the top rankers could not be induced to choose their career as a teacher in non-clinical Departments of their choice by offering them a suitable Fellowship right from their intern year. They may join the chosen department after their House Surgeoncy and be counted as one of the Junior Teachers under the designation of Demonstrators. They will concurrently work for tho post-graduate examination and take it at the end of their third year in the department. Under such an arrangement it may have to be considered whether non-clinical teachers should not be treated as a closed cadre from or to which transfers from the General Branch will ordinarily not happen. The position as regards clinical subjects is not quite so difficult and it could further be improved.

Medical Research.

It is universally emphasised that every medical college should be a Centre of Research in a small or big way depending on facilities in all its departments. No other institution can assemble the variety of disciplines available in a medical college for the conduct of research. It is a recognition of this that has caused our State Government under the able guidance and stimulus

of our Director of Medical Services to institute the Madras State Research Council to meet annually to review the research work done in the colleges during the preceding year and give approval for their continuance as also for new schemes that may be submitted. The I. C. M. R. does it for all India in a larger way. For some inexplicable reason the Colleges in South do not take a rightful share of the funds available under I. C. M. R. It appears mostly monopolised by colleges, small or big, recent or old, mostly of Northern India. Our college departments are as good if not better than the best in India, and I believe that it should be possible for us too to more fully avail of the opportunities for research afforded by the I. C. M. R. I should not be understood to say this as any criticism. I am merely stating a fact in order that it may get due consideration at appropriate hands.

My Opportunities.

During the last three years as Dean I have had opportunities and some leisure to think about the problem of medical education and I have placed a few of my thoughts before you. Conventionally I am of course no authority on medical education, as I was out in the Districts for 15 years between my first period of service of 5 years in the college and my present occupation as Dean. But then I would make this claim. I had the advantage of a spectator and observer, to watch the products of medical education. For a decade and more I have met scores of fresh men in the District, Taluk and Village Hospitals and Dispensaries who had graduated just prior to their coming to those stations. Naturally this gives a unique opportunity for me to assess the true value of the education they had received. If you permit me to share my observations with you—mark you not a criticism—they are these. They seem to lack a scientific approach to the problems of disease. Medicine may not be an exact or accurate science as Mathematics. Medicine is perhaps as much an art as a Science, consisting as it does a combination of high Physics, Chemistry, Bio-Chemistry and Bio-Physics and Biology. If the pursuit of any of these single sciences could in all conscience be difficult and exacting for a young man it will readily be seen how much more it should be to grapple with the science of medicine. This is surely not an argument for relegating medicine to the region of empiricism. Here again you will be interested to hear the Shawian view of the matter :—

Modern Graduate—Lacking in.

(1) " As a matter of fact the rank and file of doctors are no more scientific than their tailors—or if you prefer to put it the other way, their tailors are no less scientific than they.

(2) The distinction between a qualified doctor and a quack one is mainly that only the qualified doctor is authorised to sign death certificates for which both sorts seem to have equal occasion.

(3) Some of the most successful doctors on the register use quite heretical methods of treating disease and have qualified themselves solely for convenience."

If we are to live down this aspersion it is necessary for us all to practise and preach to the students that they should employ only methods of science in their day to day work, small or big. What is important for the student to grasp is that he should employ a strictly scientific approach in his day to day work. This does not mean big buildings and laboratories, though a minimum equipment such as microscope, a sterilizer, an operating table, a B. P. apparatus, ophthalmoscope, etc., are necessary for the practice of what is known as scientific medicine. Irrespective of the situation where the young graduates may work in, they should learn invariably to employ the scientific approach. The second deficiency that I have noticed is the lack of application of the principles they have been taught in the day to day conduct of their work. Again quite a few of them seem to suffer from a lack of sound grasp of basic fundamentals and an orderly and systematic mind in the examination of a case. It was also noticed that they were too ready to proceed with blunderbuss treatment with tonics and vitamins, anti-biotics, chemotherapeutic drugs before taking some little time to determine the site and nature of disease by systematic clinical examination and the facilities available with them. Another curious defect one sees in bigger hospitals is that the House Surgeon orders a multiplicity of laboratory and X-ray examinations while a simple clinical examination which he has not perhaps carefully gone through will have revealed the true diagnosis. Lastly they suffer from a lack of precision and clarity in expression as the method.

Medicine a Science.

I am offering these as no more than observations by an interested outsider and I may also say that my experience as Dean these three years confirms me in these observations. I would however add that there are scores of our young graduates who could be the best anywhere in the world, and that the bulk of them are on the whole doing a good job. I have now come to the end of my wandering and perhaps desultory talk. I have perhaps been too ambitious to cover a whole range of my experience during the last 30 years. If I have said anything which I should not I seek your indulgence. My intention and purpose is no more than to make use of this opportunity which your President and Committee have so kindly afforded me to share with you my observations on our various problems. No more authority is due to what I have put before you and I would request you to take it only in that light.

Conclusion.

Before closing I should not omit to congratulate ourselves on our excellent President Dr. Rathnavelu Subramaniam. If the Madras Medical

College and its staff and students are exceptionally lucky in having him to occupy the important chair of medicine in this great College our Association is doubly so in securing his valuable time and service in the cause of our Association. I have no doubt whatsoever that under his able guidance our Association will become more and more an instrument of service not only to its members but also in the wider context of improvement of standards of efficiency and a spirit of devoted service, to a level of pre-eminence second to none in this sub-continent.

We are fortunate in having at the helm of affairs of the Medical Department of the State a person of the eminence of Dr. V. R. Thyumanaswami. It should have been a pang to him to step aside from his Specialty to the field of Administration with which he had little to do in his earlier years of service. But this latter circumstance has instead of being a handicap, been his greatest asset. He has brought to bear upon the various problems confronting him a freshness of outlook, a deep consideration and mature judgment. He has been during the last 3 years and more indefatigable in solving the problems of medical relief, education, their expansion and improvement in various directions right from the village level to the City of Madras. His task has been no easy one and in my humble tribute to his great performance in the discharge of his onerous work I am sure all of you will join.

Thanking you all for the patient indulgence with which you have heard me, I would now close my address.